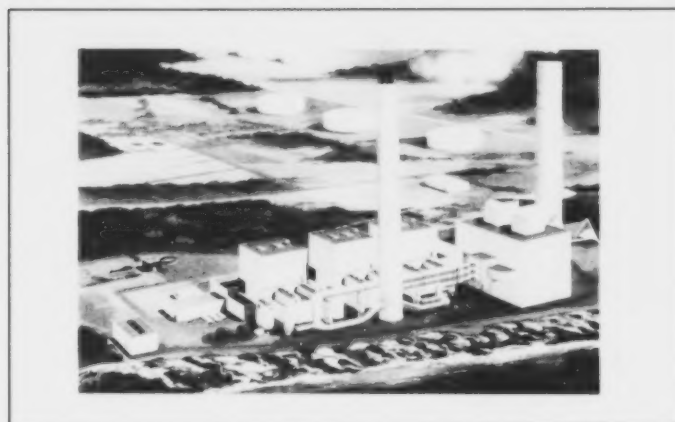
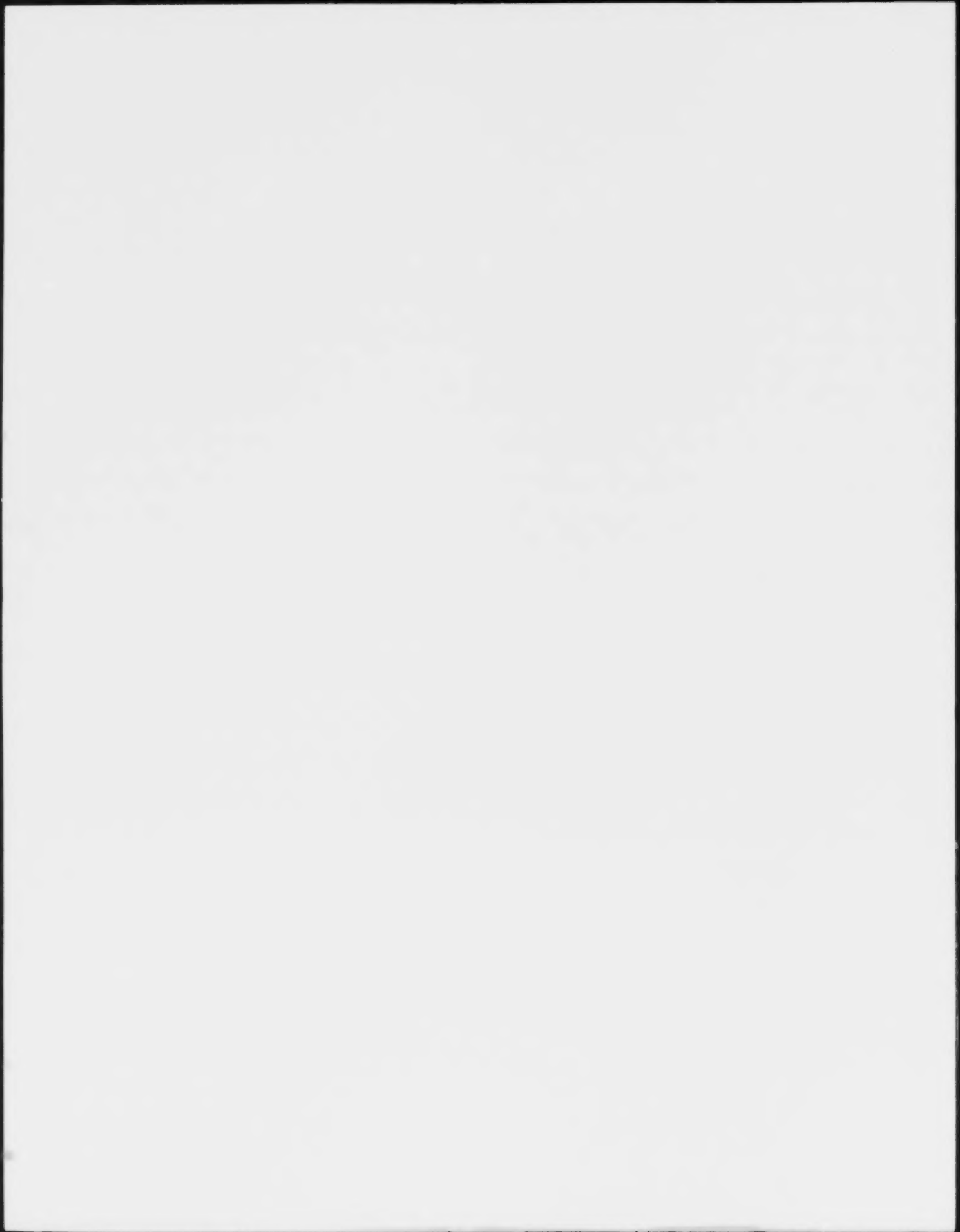


**Coleson Cove Generating Station
Refurbishment Project**
Environmental Impact Assessment
Human Health Risk Assessment Report

APRIL, 2002



Énergie NB Power





Human Health Risk Assessment Final Report
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Submitted to:

New Brunswick Power
Fredericton, New Brunswick

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AMEC Earth & Environmental
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EXECUTIVE SUMMARY

Abstract

This human health risk assessment of the Coleson Cove Generating Station (CCGS) evaluates health risks both quantitatively and qualitatively for three sets of operating conditions, in which the CCGS is assumed to use fuel oil, Orimulsion®, or natural gas as a source of fuel. Risks from criteria and noncriteria pollutants are evaluated differently. Criteria pollutants include sulphur dioxide, nitrogen oxides and respirable particulate matter. For these pollutants, extensive governmental actions have been undertaken to set concentration-based standards that are protective of health. Regulatory compliance with standards is evaluated in the Air Quality Report (JWEL, 2002). In addition to the evaluation of regulatory compliance, this risk assessment evaluates health risk for these constituents by comparing predicted annual average concentrations of these pollutants in the City of Saint John to the governmental standards.

For non-criteria pollutants, there are no governmental standards. The health risk assessment for these constituents, such as metals, is done by performing a quantitative, multiple exposure pathway risk assessment for numerous groups of people who may be exposed to them both in the air and after they have deposited on the ground. The required input parameters for such an assessment do not exist for the criteria pollutants, so they cannot be included in the multiple pathway risk assessment.

Additionally, several qualitative risk assessment evaluations were performed. Trends in air quality indicators in the study area are discussed. The health status of residents in the study area was evaluated and discussed in the context of the health status of people in the province and the country as a whole. Prevalence rates of cancer, respiratory disease, and asthma are compared for the study area, the province, and the nation. The current scientific literature regarding the causation of asthma is discussed. Also, recent scientific studies showing associations between air pollution and cardiorespiratory disease have been summarized and their relevance to evaluating the impacts of emissions from the CCGS are discussed. These studies have been performed in Saint John, elsewhere in Canada, and in the United States, and they form the basis of the regulatory actions both in Canada and the United States that have resulted in the recent issuance of standards for fine respiratory particulate matter.

Estimated impacts from the CCGS are also compared to background conditions for metals (when existing data allow) to determine if the facility is a major source of these metals in the environment in the City of Saint John.

Overall conclusions of the health risk assessment are that facility emissions under all three fueling scenarios cause no exceedances of governmental standards or levels of concern for carcinogenic and noncarcinogenic risk. Facility emissions under all three fueling scenarios are low compared to background air pollutant concentrations for criteria pollutants and background risk levels for non-criteria pollutants, indicating that the CCGS is not a major cause of air pollution or adverse health conditions in the City of Saint John. Emissions of environmental concentrations of all evaluated constituents will be lower assuming that the proposed refurbishment project is

completed. Estimated carcinogenic and noncarcinogenic risks will also be lower, assuming the refurbishment is completed. Thus, the refurbishment project will confer health benefits. However, these reductions are small and may not result in a measurable decrease in hospitalizations, diagnosed cases of asthma, or identified cases of cancer within the City of Saint John or measurable decreases in criteria pollutant concentrations.

Introduction

This document presents a Multipathway Human Health Risk Assessment of the Coleson Cove Generating Station in Saint John County, New Brunswick, operated by New Brunswick Power (NB Power). The Coleson Cove Generating Station currently generates electricity by burning heavy fuel oil (fuel oil #6) as an energy source. This comprehensive risk assessment estimates potential risks to human health associated with direct and indirect exposure to emissions from the Coleson Cove Generating Station under three sets of conditions:

1. The Existing Condition - Fuel Oil #6 - High Load Scenario, in which the Facility continues to use heavy fuel oil as an energy source;
2. The Refurbishment Project - Orimulsion® - High Load Scenario, in which the Facility is assumed to use Orimulsion® as an energy source; and
3. The Natural Gas - High Load Scenario, in which the Facility is assumed to use natural gas as an energy source.

Unlike the Air Quality Report (JWEL, 2002), which evaluates the facility operating at low, medium, and high capacity factors for each fueling scenario, the health risk assessment evaluates risks solely assuming the maximum capacity factor. This is done intentionally to ensure that health risks are overestimated and the results are health protective. The term "high load scenario" as used in this report means that it is assumed that the CCGS operates 24 hours a day, 365 days a year at maximum capacity. Actual operating conditions are, of course, lower because of power demand requirements and the need for periodic maintenance activities.

Estimates of the potential carcinogenic risks (that is, the chance of developing cancer) and noncarcinogenic risks (that is, the likelihood that toxic health effects other than cancer may occur) are made and presented for several types of exposures to emissions from the facility.

The risk assessment guidance requires that risks be evaluated for three groups of people (residents, farmers, and fishers) who may or may not actually live in the study area around the facility that is the subject of the risk assessment. This is required so that all of the ways that facility emissions could affect human health are evaluated. The guidance requires that these groups of people are assumed to be located in areas defined as locations of maximum impact where it is reasonable to anticipate that they could be exposed to facility emissions.

After evaluating the results of the air dispersion and deposition modeling, AMEC identified a location directly adjacent to the property boundary as the location for the maximally affected hypothetical resident (a recreational fisher) and a location northwest of the City of Saint John as the location for the maximally affected hypothetical farmer. In addition, the City of Saint John was

identified as another location for which risks would be evaluated for residents and recreational fishers. Each of these groups of people are called "receptors" in this risk assessment. The five receptors identified are summarized as follows:

- resident living at the hypothetical residential area most affected by the facility;
- resident living in the City of Saint John;
- resident living at the hypothetical residential area most affected by the facility who fishes recreationally;
- resident living in the City of Saint John who fishes recreationally; and
- farmer living at the farm most affected by the facility.

For each of these receptors, both adults and children were separately evaluated. It was assumed that each of the receptors could be exposed through various direct pathways (such as inhalation of air containing emissions from the facility) and indirect pathways (such as consumption of food products or water that have been exposed to facility emissions).

Exposures to facility emissions were calculated using numerous conservative (upper-bound) exposure assumptions that tend to overstate potential risks. Therefore, the potential risks calculated in this risk assessment do not represent actual risks to people living near the Coleson Cove Generating Station, but rather represent the reasonable upper-bound estimates of risk to hypothetical receptors assumed to engage in all activities that may result in exposures to facility emissions.

Risk Assessment Methodology

This risk assessment followed the four step method developed by the National Research Council of the U.S. National Academy of Sciences and routinely used by Canadian regulatory agencies. The four steps are:

1. Hazard Identification;
2. Toxicity Assessment;
3. Exposure Assessment; and
4. Risk Characterization.

The methods and results of each of the four steps of the risk assessment are summarized in the following paragraphs. The methodology used to estimate potential exposures and risks is consistent with guidance from U.S. EPA (1998a,b, 1999, 2000).

Hazard Identification

The compounds to be quantitatively assessed in the risk assessment are selected in the Hazard Identification step. The compounds expected to be emitted from the Coleson Cove Generating Station depend upon the type of fuel used at the facility. Compounds of potential concern were identified for each set of operating conditions based upon measurements of compounds in fly ash from emissions from the CCGS, measurements of compounds in fly ash from emissions from

similar facilities, measurements of compounds in fly ash from emissions from a facility using Orimulsion® as fuel, U.S. EPA sources, and other literature sources. Each compound expected to be emitted from the facility was evaluated for its potential to pose a threat to human health. The factors taken into consideration in this process are the maximum amount of the compound expected to be released, its toxicity, and its mobility and persistence in the environment. A subset of compounds was then selected for inclusion in the quantitative risk assessment. An average emission rate was then estimated for each of these compounds based on fly ash measurements, facility emission measurements, engineering calculations, air pollution control equipment specifications, U.S. EPA AP-42 default emission factor tables, and/or information gathered from the scientific literature.

In order to estimate air concentrations of the compounds of potential concern in the region around the CCGS, air dispersion modeling was conducted using standard, conservative models in accordance with standard U.S. EPA guidelines, which are routinely used by Canadian regulatory agencies. These models take into account emission rates, physical properties of the emitted compounds and local weather data to predict the 5-year average air concentrations. The results were used to provide the air concentration data used to evaluate the various receptor locations.

Deposition modeling was performed using the results of the air dispersion modeling and meteorological data to describe the behavior of compounds associated with particles in the air that deposit on soils or water bodies. Additional modeling was performed to predict compound concentrations in soils and surface waters resulting from deposition, runoff, and other environmental fate and transport processes.

Toxicity Assessment

In order to determine if exposures to the compounds of potential concern may potentially result in adverse human health effects, it is necessary to have a numerical estimate of the toxicity of each compound of potential concern. The toxicity assessment identifies the types of adverse health effects a compound of potential concern may potentially cause and defines the relationship between the dose of a compound and the likelihood or magnitude of an adverse effect (dose-response). Adverse effects are characterized as carcinogenic or noncarcinogenic. Dose-response relationships are defined for oral exposure and for exposure by inhalation.

Noncarcinogenic effects are those other than the potential to cause cancer. They encompass a wide variety of effects that range from body weight changes to more serious effects such as liver or kidney toxicity. Compounds with noncarcinogenic effects are assumed by regulatory agencies to have a dose, called the threshold, below which no adverse effect occurs or, conversely, above which an adverse effect may be seen. Noncarcinogenic dose-response values define a safe level of exposure for humans. The incorporation of safety factors into the derivation of the safe level provides reasonable certainty that the safe dose is below the threshold dose and that no noncarcinogenic health effects are expected to occur, even if daily exposure to that dose were to occur for a lifetime to especially sensitive members of the population.

The conservative underlying assumption of regulatory risk assessment for compounds with known or suspected potential carcinogenic effects is that no threshold exists. In other words, it is assumed that a finite level of risk is associated with any dose above zero. The regulatory agencies have developed computerized models that estimate the carcinogenic potency of a compound. This numerical estimate is referred to as a cancer slope factor.

In this risk assessment, the relationship between the dose of a compound and the likelihood and magnitude of an adverse effect was determined for each compound included in the quantitative risk assessment. Both potential carcinogenic and potential noncarcinogenic effects were considered in the risk assessment. Dose-response information used in the risk assessment was identified from Canadian and U.S. government sources.

Exposure Assessment

The results of the various modeling tasks performed in the Hazard Identification step provide estimates of the concentrations of the compounds of potential concern in air, soil, surface water, and food products as a result of facility emissions. These concentrations are used to predict receptor exposure to the compounds.

To estimate potential human exposure to compounds of potential concern, potential exposure pathways were defined for each receptor. Potential exposure pathways describe ways in which receptors may be exposed to compounds of potential concern in the various environmental media.

For each of the receptors identified in this risk assessment, it was assumed that exposure could occur through the following direct and indirect pathways:

- inhalation of emissions from the facility;
- incidental ingestion of soil while working or playing outdoors;
- ingestion of produce grown in backyard gardens or farms;
- ingestion of fish caught from Spruce Lake, Ludgate Lake, and Menzies Lake;
- consumption of food products (beef, dairy, pork, poultry, and eggs) from a local farm; and
- drinking water exposure to water from the Spruce Lake Reservoir.

For each fueling scenario, compound concentrations present at each receptor location (hypothetical maximum residence, City of Saint John, farm, and Lakes) were estimated using conservative (health-protective) assumptions. In addition, conservative assumptions were also made about the magnitude of each exposure, such as the quantity of soil or fish that is ingested on a daily basis. The use of many upper-bound factors in calculating potential risks results in an estimate that tends to overstate actual risks, if any, which may occur as a result of receptors' potential exposure to facility emissions.

Risk Characterization

The potential exposure dose for each receptor from each compound via each route of exposure was combined with the appropriate dose-response value for that compound in order to estimate the potential for adverse health effects.

In the risk characterization process, to evaluate if potential noncarcinogenic effects may occur due to exposure to compounds of potential concern, the ratio of the receptor's exposure dose to the noncarcinogenic dose-response value is calculated for each compound of potential concern. This ratio is termed the hazard quotient. If the hazard quotient ratio is less than one, no adverse noncarcinogenic health effects are expected to occur as a result of exposure to that compound via that route of exposure. The hazard quotient for each pathway by which each receptor is assumed to be exposed are summed to yield a hazard index for that compound. A total hazard index is then calculated for each receptor by summing the compound-specific hazard indices for that receptor for each toxicity endpoint for which it is appropriate to sum the effects of several compounds of potential concern. A total hazard index of less than one for a given receptor for each toxicity endpoint provides further evidence that no adverse noncarcinogenic health effects are expected to occur as a result of that receptor's potential exposure to environmental media given the emission rates and exposures assumed in the risk assessment. As a screening step, hazard indices for all chemicals are summed to determine if it is necessary to calculate toxicity endpoint-specific hazard indices. If the total hazard index is less than one, then hazard indices for each toxicity endpoint are not necessary.

The purpose of carcinogenic risk characterization is to estimate the likelihood, over and above the background cancer rate, that a receptor will develop cancer in his or her lifetime as a result of exposures to compounds of potential concern in environmental media due to facility emissions. The product of the exposure dose of a compound via a particular pathway times the cancer slope factor for that compound is a unitless value. It provides an estimate of the potential carcinogenic risk associated with a receptor's exposure to that compound via that pathway. This value is termed the excess lifetime cancer risk. The excess lifetime cancer risk for each compound is calculated by summing the excess lifetime cancer risk values for each pathway by which the receptor is assumed to be exposed. The total excess lifetime cancer risk for each receptor is calculated by summing the compound- and pathway-specific excess lifetime cancer risks. Excess lifetime cancer risks are compared to a target risk of 1×10^{-5} , or one excess cancer case per 100,000 people exposed. An excess lifetime cancer risk for a compound that is less than the target risk of 1×10^{-5} is less than the regulatory level of concern. A total excess lifetime cancer risk for a receptor that is less than the target risk of 1×10^{-5} provides further evidence that the receptor's potential exposure to facility emissions will not result in unacceptable cancer risk.

For each of the three fueling scenarios, the compound-specific hazard index for each compound of potential concern is below 1 for each receptor. The total hazard index for all compounds of potential concern is also below 1 for each receptor, and the toxicity endpoint-specific hazard index is also below 1 for each receptor. The compound-specific carcinogenic risk for each compound of potential concern is less than 1×10^{-5} for each receptor. The total carcinogenic risk for all compounds of potential concern is less than 1×10^{-5} for each receptor.

The following table shows the estimated total excess lifetime cancer risk and total hazard index for each receptor evaluated for each operating scenario.

SUMMARY OF CARCINOGENIC AND NONCARCINOGENIC RISK ESTIMATES FOR NON-CRITERIA POLLUTANTS

Scenario	Receptor	Total Excess Lifetime Cancer Risk	Total Hazard Index*
Existing Condition – Fuel Oil #6 – High Load Scenario	Maximum Location Child Resident	7×10^{-8}	0.17*
	Maximum Location Adult Resident	1×10^{-7}	0.08
	Saint John Child Resident	7×10^{-8}	0.08*
	Saint John Adult Resident	2×10^{-7}	0.04
	Maximum Location Child Fisher	8×10^{-8}	0.18*
	Maximum Location Adult Fisher	2×10^{-7}	0.12*
	Saint John Child Fisher	8×10^{-8}	0.09*
	Saint John Adult Fisher	3×10^{-7}	0.12*
	Child Farmer	6×10^{-8}	0.08*
	Adult Farmer	1×10^{-7}	0.04
Refurbishment Project – Orimulsion® – High Load Scenario	Maximum Location Child Resident	7×10^{-9}	0.02
	Maximum Location Adult Resident	1×10^{-8}	0.009
	Saint John Child Resident	1×10^{-8}	0.01
	Saint John Adult Resident	5×10^{-8}	0.007
	Maximum Location Child Fisher	8×10^{-9}	0.04
	Maximum Location Adult Fisher	2×10^{-8}	0.03
	Saint John Child Fisher	2×10^{-8}	0.03
	Saint John Adult Fisher	6×10^{-8}	0.03
	Child Farmer	1×10^{-8}	0.01
	Adult Farmer	4×10^{-8}	0.006
Natural Gas – High Load Scenario	Maximum Location Child Resident	2×10^{-10}	5×10^{-6}
	Maximum Location Adult Resident	4×10^{-10}	1×10^{-6}
	Saint John Child Resident	8×10^{-11}	1×10^{-6}
	Saint John Adult Resident	3×10^{-10}	5×10^{-7}
	Maximum Location Child Fisher	6×10^{-10}	3×10^{-4}
	Maximum Location Adult Fisher	3×10^{-9}	3×10^{-4}
	Saint John Child Fisher	4×10^{-10}	3×10^{-4}
	Saint John Adult Fisher	3×10^{-9}	3×10^{-4}
	Child Farmer	1×10^{-9}	2×10^{-6}
	Adult Farmer	3×10^{-9}	1×10^{-6}

*Footnoted result is the maximum toxicity endpoint-specific hazard index.

Cancer risks estimated by this risk assessment for all three fueling scenarios are all so low that they are immeasurable and can be considered to be indistinguishable from each other. Although the numerical estimates can be shown in the various tables to be different numbers, some of which are lower than others, they are so low that any differences between them have limited, if any, significance. According to the Atlantic Health Sciences Corporation (2001), the population of the City of Saint John in 2000 was 72,269. The estimated excess lifetime cancer risk from this risk assessment that would be equivalent to one additional case of cancer in Saint John over a year is 4.1×10^{-4} . The estimated excess lifetime cancer risk from this risk assessment that would be equivalent to one additional case of cancer in Saint John over the course of a 30-year period is 1.4×10^{-5} . Clearly, any estimated risk level that is less than 4.1×10^{-4} is not measurable over a year, and any risk level that is less than 1.4×10^{-5} is not measurable over 30 years.

According to Atlantic Health Sciences Corporation (2001), cancer incidence rates for the City of Saint John were 218 cases (total cancers) in 2001 in males and 195 cases (total cancers) in females in 2001. The total number of cases was thus 413 out of a population of 72,269. If the estimated excess lifetime cancer risk from this risk assessment for any fueling scenario was 4.1×10^{-4} or higher, then one would predict that this number of cancer cases would increase from 413 to 414 cases in the population in a year. However, no estimated lifetime cancer risk was higher than 3×10^{-7} . Thus, the predicted increase in cancer cases per year would be from 413 cases to 413.0007 cases. This increase in risk is not measurable.

Over the course of the assumed period of operation of 30 years, there will be more cases of cancer in Saint John due to the many causes of cancer. If it is assumed that the cancer incidence rate due to all sources remains constant and the population remains constant, one can predict that the number of cases of cancer over 30 years will be $413 \times 30 = 12,390$. If the estimated excess lifetime cancer risk from this risk assessment for any fueling scenario was 1.4×10^{-5} or higher, then one would predict that this number of cancer cases would increase from 12,390 to 12,391 cases in the population over the course of 30 years. Such a risk, although small, would be measurable. However, no estimated lifetime cancer risk was higher than 3×10^{-7} . Thus, the predicted increase in cancer cases over 30 years would be from 12,390 cases to 12,390.02 cases. This increase in risk is also not measurable.

Noncancer risks estimated by this risk assessment for all three fueling scenarios are all so low that they are indistinguishable from each other. Although the numerical estimates for hazard quotients and hazard indices can be shown in the various tables to be different numbers, some of which are lower than others, they are so low that any differences between them have limited, if any significance.

All tolerable daily intakes and reference doses, which are levels of a chemical to which a person can be exposed every day for their entire lifetime without experiencing an adverse health effect, are derived with many safety factors. Thus, a hazard index over 1.0 would exceed a regulatory level of concern, but it would not exceed a level at which adverse effects have ever been demonstrated in either animals or humans.

For instance, the arsenic hazard quotient for the fisher child receptor at the maximum hypothetical residential location for the Existing Condition – Fuel Oil #6 – High Load Scenario is 0.002. This value is 500 times lower than the tolerable daily intake. However, the tolerable daily intake was derived from a no effect level in humans divided by an additional safety factor of 3. The estimated dose for this receptor is, in fact, 25,000 times lower than the lowest dose level that was shown to cause adverse effects on the skin of humans exposed to arsenic in drinking water.

Similarly, the vanadium hazard quotient for this receptor is 0.18. This value is 6 times lower than the tolerable daily intake. However, the tolerable daily intake was defined as 100 times lower than the lowest dose in an animal experiment where no adverse effects were seen, even in the animals given a 10-fold higher dose of vanadium. The estimated dose to the fisher child at this location is, in fact, over 5,000 times lower than the dose level in the animal experiment where no adverse effects were seen.

As well, the mercury hazard quotient for the child fisher receptor at this location is 0.09. This value is 11 times lower than the tolerable daily intake. However, the tolerable daily intake was defined as 1,000 times lower than the lowest dose in an animal experiment that resulted in adverse effects on the animals. The estimated dose to the fisher child is, in fact, over 10,000 times lower than the dose level in the animal experiment that caused adverse effects.

The tolerable daily intakes and reference doses are defined by regulatory agencies to be intentionally very health-protective. The agencies assume that all dose levels within 10 fold of an effect level could also have adverse effects. They assume that humans are more sensitive to the chemical compared to animals in all cases, by a factor of 10. They always assume that some sensitive human subpopulations are 10-fold more sensitive than other humans. All of these assumptions are made so that estimated doses of chemicals from risk assessments that yield hazard quotient estimates less than 1.0 are certain to be totally devoid of risk. In addition, if the hazard quotient is less than 1.0, the additional margin of safety addresses the concern that is sometimes raised that people may be exposed to background levels of the chemical from other sources. In this case, the highest toxicity endpoint-specific hazard index is 0.18, which means that even if background exposures were five times higher than the facility-related exposures, the regulatory level of concern hazard index of 1 would still not be exceeded. Thus, all hazard index estimates from this risk assessment are essentially indistinguishable. In all cases, there is a high degree of certainty that no adverse effects will occur.

For instance, the hazard index for all chemicals combined for the resident child assumed to live in Saint John for the Refurbishment Project – Orimulsion® – High Load Scenario is 0.01. This is 100 times lower than the noncancer benchmark of 1. No endpoint specific hazard index exceeds 0.008, which is 125 times lower than the benchmark of 1. In this case, there is a high degree of certainty that no adverse effects will occur. For the same receptor, the Natural Gas - High Load Scenario gives a total hazard index summed over all chemicals of 1×10^{-6} , which is far lower than the benchmark of 1, but these two results are, in fact, indistinguishable given the manner in which tolerable daily intakes are derived. Noncancer risk is inversely proportional to hazard index as the hazard index drops from high values down to the benchmark of 1.0. However, once the estimated

hazard index drops to levels below the benchmark level of concern, the differences in the hazard index are no longer distinguishable from one another.

In conclusion, for both carcinogenic and noncarcinogenic risk, the risks for all three scenarios are low enough that they should be viewed as essentially indistinguishable from each other in terms of a direct comparison to regulatory decision-making criteria. Reducing risks to levels below regulatory levels of concern can be viewed as adding societal benefits, to a point, because reduction of carcinogenic and noncarcinogenic risks to levels beyond required regulatory levels can decrease the uncertainty that is inherent in the risk estimation process. Thus, if one scenario has an estimated risk that is ten-fold below the regulatory level of concern, there is a societal benefit to choosing a second scenario that has an estimated risk that is 100 times below the regulatory benchmark, because one has increased certainty that if important factors were inadvertently left out or underestimated, the true result is more likely to still be acceptable if the margin of safety is greater for the second case. However, this increase in certainty cannot be extended *ad infinitum*. At some point reasonable people who evaluate the conservativeness of the risk assessment process, which has many safety factors inherently built into it, will have to conclude that an *additional* safety factor is simply not necessary. By this logic, one can conclude that reducing cancer risk from 1×10^{-7} to 1×10^{-8} can be viewed as having a societal benefit given a regulatory level of concern of 1×10^{-5} , but reducing it to 1×10^{-10} provides no tangible benefit.

This is the situation with the three fueling scenarios. Reducing the estimated cancer risk from 0.03 cases in 100,000 for the Existing Condition - Fuel Oil #6 - High Load scenario to 0.006 cases in 100,000 for the Refurbishment Project - Orimulsion® - High Load Scenario can be viewed as conferring a societal benefit in terms of reducing any uncertainty inherent in the risk assessment. In the first case, the additional margin of safety (over and above the margins of safety that are built into the risk assessment process) that the regulatory goal was met was 33-fold. In the second case, the additional margin of safety is 166-fold. Further reducing the estimated cancer risk to 0.0003 cases in 100,000 for the Natural Gas - High Load Scenario may indeed increase the margin of safety further, but attaining this additional reduction in risks does not equate to a proportionally increased benefit, because the result is already so far beyond the regulatory goal.

Risk Characterization of Criteria Pollutants

Estimated annual average concentrations of sulphur dioxide, nitrogen oxides, and respirable particulate matter were compared to the measured levels in Saint John and to the governmental standards. The CCGS is not a major source of sulphur dioxide, nitrogen dioxide, PM_{10} , or $PM_{2.5}$ in the City of Saint John, nor would it be if the refurbishment project were executed. Neither estimated concentrations resulting from the CCGS nor the existing ambient concentrations that result primarily from sources other than the CCGS exceed standards derived to protect human health. Nonetheless, the CCGS may be responsible for a fraction of the ambient concentrations in the City of Saint John. If the refurbishment project were executed, the contribution of the CCGS to the concentrations in Saint John would drop, so the ambient concentrations in Saint John would also drop assuming that no other sources were increased. This estimated drop the concentrations of criteria pollutants must be viewed as a health benefit.

However, since the CCGS is not a major source of these criteria pollutants in the City of Saint John, the predicted decreases in concentrations of criteria pollutants are modest and may be difficult to measure. For instance, assuming that the average concentration of sulphur dioxide in Saint John is 18 micrograms per cubic meter of air ($\mu\text{g}/\text{m}^3$), which is the average of the Forest Hills and the Prince William Street measurements from 1995-2000, the average concentration is estimated to drop from 18 to $17.5 \mu\text{g}/\text{m}^3$. The estimated additional improvement in air quality assuming that the CCGS used natural gas as fuel would result in an estimated concentration of $16.5 \mu\text{g}/\text{m}^3$. 256

Similarly, assuming that the average concentration of nitrogen oxides in Saint John is $14.5 \mu\text{g}/\text{m}^3$, which is the average of the Forest Hills and the Prince William Street measurements from 1995-2000, the average concentration is estimated to drop from 14.5 to $14.4 \mu\text{g}/\text{m}^3$. The estimated additional improvement in air quality assuming that the CCGS used natural gas as fuel would result in an estimated concentration of $14.2 \mu\text{g}/\text{m}^3$. For respirable particulate matter, the CCGS is estimated to contribute only 0.1% to the concentrations of PM_{10} and $\text{PM}_{2.5}$ in the City of Saint John. Thus, the estimated improvements in the concentrations assuming that the refurbishment project is executed are probably not measurable.

Qualitative Risk Characterization

The risk assessment has reviewed and evaluated data and information on the health status of people residing in the study area and the recent scientific research showing associations between air pollution and cardiorespiratory diseases. It was found that lung cancer appears to be elevated in the City of Saint John compared to the province and the nation, but lung cancer rates are lower in the Saint John area compared to the province and the nation. Asthma does not appear to be elevated in Saint John. The status of other respiratory diseases is unclear, because various statistical measures are not consistent. Smoking rates have been shown in a variety of statistical sources to be elevated in Saint John compared to the province and the nation, and this information should be considered when cancer rates are evaluated. 22

Several epidemiology studies have been performed in Saint John to determine if the variability in adverse respiratory or cardiac health effects might be associated with variability in air pollutant concentrations. In one study, asthma emergency department admissions were found to be statistically significantly associated with ozone levels. No effects of particulate matter were shown. In another, the number of emergency department admissions for respiratory diseases was shown to be associated with smoking and viral upper respiratory tract infections. Lastly, other researchers studied emergency department admissions for respiratory and cardiac diseases and found that ozone and sulphur dioxide could explain the variability in admissions. No effects were seen for respiratory particulate matter in multipollutant analyses. Fungal spores were also associated with adverse health effects during warm months.

In conclusion, the studies performed in Saint John provide little support for the hypothesis that the variability in the rates of adverse health outcomes is attributable to the variability in the concentration of respiratory particulate matter, such as PM_{10} or $\text{PM}_{2.5}$. Studies performed elsewhere in Canada and the United States have shown that part of the variability in health

outcome rates may be associated with varying rates of particulate matter concentrations, but the Saint John studies differ in their conclusions from these other studies. The conclusions of the Saint John studies point to ozone, sulphur dioxide, fungal spores, and smoking as potential causes of respiratory and cardiac disease.

However, based on a review of all of the studies performed in Canada and the United States, it can be stated that reductions in ambient concentrations of one or more of the following air pollutants (ozone, sulphur dioxide, nitrogen dioxide, carbon monoxide, and respirable particulate matter [$PM_{2.5}$ and PM_{10}]) could be expected to confer health benefits to residents of Saint John. These benefits would be associated with decreases in hospitalizations for asthma and other respiratory diseases, hospitalizations for cardiac diseases, and mortality from cardiorespiratory diseases.

Even though the decrease in air pollutant concentrations that are estimated to occur when the Coleson Cove Generating Station is refurbished are expected to have a beneficial effect on human health, there is no way to quantitatively predict the number of cases of each health outcome that will be prevented. This human health risk assessment has predicted the differences in carcinogenic and noncarcinogenic health risk for selected metals, polycyclic aromatic hydrocarbons, and volatile organic chemicals, because there are standard risk assessment methodologies developed by government health protection agencies to do so. Such methods have been widely used for environmental decision making for over 25 years. However, no such methods exist for estimating the number of hospitalizations for asthma, chronic obstructive pulmonary disease, or cardiovascular disease or other similar health outcomes. Based on the accompanying review of recent scientific studies, one can make only qualitative conclusions about the improvement of the health status of residents of Saint John expected with the refurbishment.

In addition, although it may be reasonable to assume that some asthma and cardiorespiratory symptoms and disease outcomes are caused by air pollution, these diseases are multi-causal. Asthma researchers believe that exposure to specific allergens, such as those associated with pollens, fungi, dust mites, insects and pet dander, and environmental tobacco smoke are the major causes of asthma. Of course, outdoor air pollution is recognized to exacerbate asthma symptoms, as are other environmental factors, such as exposure to fragrances and household cleaning products, viral infections, exercise, and cold air. Similarly, chronic obstructive pulmonary disease is associated with smoking in addition to air pollution. Thus, reduction of emissions of sulphur dioxide, nitrogen dioxide, respirable particulate matter, and specific chemicals associated with the particulate matter will reduce the concentrations of some, but not all, of the agents that can cause or exacerbate cardiorespiratory diseases.

This must be viewed as a benefit to the public health. However, the causation of these diseases and the triggering of their acute symptoms are extremely complex phenomena that cannot be explained in totality. Thus, decreases in the emissions from Coleson Cove Generating Station may or may not translate into a measurable decrease in the number of new cases of cardiorespiratory diseases, the number of cardiorespiratory hospitalizations, the number of asthma attacks in children, or any other such similar health outcomes. The causation of these



diseases is too complex to make any such quantitative predictions. Nonetheless, reductions in the emissions of respirable particulate matter and other criteria pollutants is a public health benefit.

1.0 INTRODUCTION

This document presents a Human Health Risk Assessment of the Coleson Cove Generating Station (CCGS), Lorneville, New Brunswick, under three sets of assumed operating conditions, each using a different type of fuel. The CCGS is operated by New Brunswick Power Corporation (NB Power). The risk assessment was conducted to support the Environmental Impact Assessment for the Coleson Cove Refurbishment Project (CCRP), which proposes to change the fuel used in plant operation from heavy fuel oil (fuel oil #6) to a fuel called Orimulsion®. The facility location and surrounding area are depicted in Figure 1-1.

This document has evaluated risks resulting from exposure to stack emissions from the CCGS under three sets of assumed operating conditions:

- **Existing Operation - Fuel Oil #6 - High Load Scenario**, based on facility operating conditions in which the facility uses heavy fuel oil as a fuel source and is continuously operating at maximum capacity;
- **Refurbishment Project - Orimulsion® - High Load Scenario**, in which the facility uses Orimulsion® as a fuel source and is continuously operating at maximum capacity; and
- **Natural Gas - High Load Scenario**, in which the facility uses natural gas as a fuel source and is continuously operating at maximum capacity.

Risks were estimated for key receptors who are potentially exposed to emissions from the facility. Because guidance on conducting risk assessments of stack emissions is not available from Canadian regulatory authorities, the risk assessment uses state-of-the-art methodologies that are consistent with guidance from U.S. EPA (1998a,b; 1999b, 2000).

1.1 Project Summary

This risk assessment estimates potential health risks associated with stack emissions from the CCGS under Fuel Oil, Orimulsion®, and Natural Gas Conditions. The emission source parameters used in this assessment assume that the facility is operational 24 hours per day, 365 days per year for 30 years at maximum capacity. Compound-specific emission rates for the three sets of operating conditions have been estimated using various information sources, including provincial regulatory requirements, measurements of constituents in fly ash from the CCGS and other currently operational combustion facilities equipped with similar air pollution control technologies, other information prepared for the review and permitting of other combustion facilities, and engineering calculations.

The compounds evaluated in the risk assessment are defined in the Hazard Identification process. Environmental concentrations of these compounds are estimated for the CCGS using atmospheric dispersion and deposition modeling and fate and transport modeling techniques. These concentrations are combined with receptor-specific intakes to determine potential risks to human health.

As shown in Figure 1-1, the CCGS is located in Lorneville, New Brunswick, about 15 km southwest of the City of Saint John, immediately inland from the Bay of Fundy.

In the Existing Operation - Fuel Oil #6 - High Load Scenario at CCGS, heavy fuel oil (also referred to as fuel oil #6) is used to produce power in three generating units, producing up to 350 megawatts (MW) in each unit, for a total of 1050 MW, at full load. At any one point in time the load may vary, with an objective of running any one unit at a reasonably high load to meet the current power demands. The exhaust gases from each unit will be directed to one of three flues in the main stack and are released to the environment from the flue exit at the top of the stack. The existing stack contains three flues, one for each generating unit, and there is no direct connection between the flues in the stack.

In the the Refurbishment Project - Orimulsion® - High Load Scenario at CCGS, it is proposed that Orimulsion® will be used to produce power in three generating units, producing up to 350 MW in each unit, for a total of 1050 MW, at full load. At any one point in time, the load may vary, with an objective of running any one unit at a reasonably high load to meet the current power demands. The exhaust gases from all three units will be directed to the emission control equipment, dry electrostatic precipitators and wet electrostatic precipitators (ESPs) and then to the two proposed Flue Gas De-sulphurization (FGD) systems, each capable of handling exhaust gases associated with 80- 525 MW of power generation. The exhaust gases from each FGD system will be directed to the new stack, and then released to the environment from the flue exit at the top of the stack. The new stack will contain two flues, one for each FGD system, and there will be no direct connection between the flues in the stack.

In the the Natural Gas - High Load Scenario, the facility is assumed to use natural gas to produce power in three generating units, producing up to 350 megawatts (MW) in each unit, for a total of 1050 MW, at full load. At any one point in time the load may vary, with an objective of running any one unit at a reasonably high load to meet the current power demands. The exhaust gases from each unit are assumed to be directed to one of three flues in the main stack and then released to the environment from the flue exit at the top of the stack. The existing stack contains three flues, one for each generating unit, with no direct connection between the flues in the stack.

1.2 Risk Assessment Approach

The approach adopted in this risk assessment is consistent with the approach recommended by the United States' National Research Council. The NRC, established by the National Academy of Sciences to further scientific knowledge and to advise the U.S. Federal government, has established a four-step paradigm for conducting health-based risk assessments (NRC, 1983). This paradigm has been adopted by U.S. EPA as well as Canadian national and provincial regulatory agencies. In accordance with the NRC recommendations, this risk assessment is organized into the following four steps:

- Hazard Identification;
- Toxicity Assessment;

- Exposure Assessment; and
- Risk Characterization.

Each of these steps is described below.

Hazard Identification

In this step, compounds which are anticipated to be emitted from the CCGS under the three sets of operating conditions are identified. From the list of compounds expected to be emitted, those compounds which are expected to be the most toxic, mobile, persistent and/or prevalent in the environment are selected for inclusion in the quantitative risk assessment. These compounds are designated as Compounds of Potential Concern (CPC). The rates at which these compounds are currently or are predicted to be emitted from the facility are then estimated for each set of operating conditions.

Toxicity Assessment

The purpose of the Toxicity Assessment is to determine the relationship between the magnitude of exposure for each CPC (dose) and the occurrence of specific health effects for a receptor (response). This risk assessment includes an evaluation of both potentially carcinogenic and potentially noncarcinogenic effects. Dose-response criteria were obtained from Health Canada (1996), the Canadian Ministries of the Environment (1999) or from the Atlantic PIRI (1999). If these sources provided no dose-response criteria for a CPC, criteria were obtained from U.S. Environmental Protection Agency (U.S. EPA) sources. If no dose-response information was available for a given CPC, then criteria for a structurally-related compound was substituted.

Exposure Assessment

In the Exposure Assessment, the magnitude and frequency of receptors' exposure to CPCs is quantified. Potential human receptors are identified based on characteristics of the site and surrounding area. All relevant indirect and direct routes of exposure to environmental media are examined for each receptor.

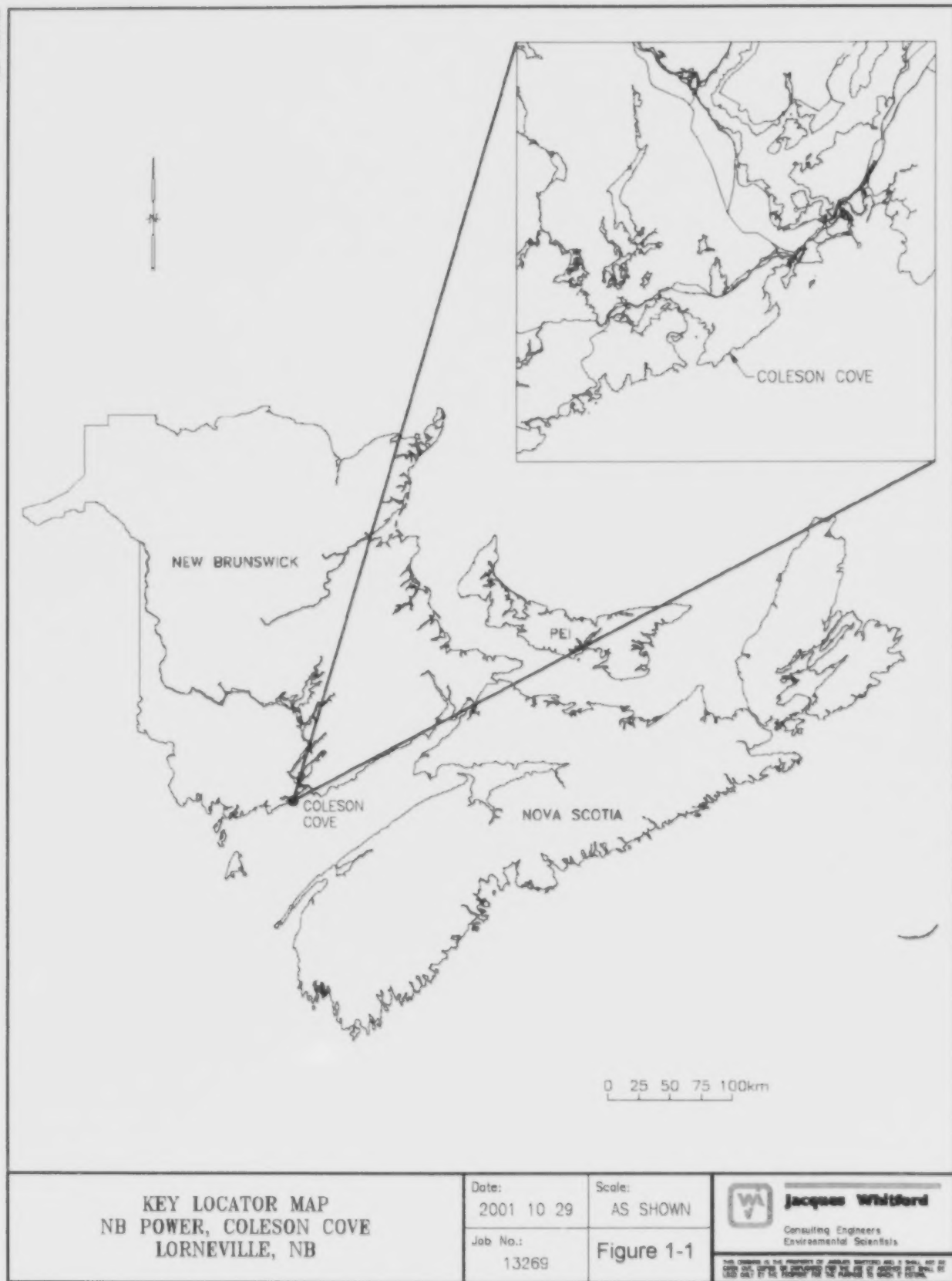
Risk Characterization

The Risk Characterization combines the results of the Exposure Assessment with the results of the Toxicity Assessment to derive quantitative estimates of the potential for adverse health effects to occur as a result of potential exposure to predicted emissions from the CCGS. For each set of operating conditions, the potential for both noncarcinogenic and carcinogenic effects is estimated for each receptor for each potential exposure pathway identified in the Exposure Assessment. The Risk Characterization also includes a discussion of the potential for respiratory health effects as a result of emissions from the facility.



1.3 Organization of Report

The Human Health Risk Assessment for the CCRP is organized as follows: Section 2.0 presents the Hazard Identification. The Toxicity Assessment is described in Section 3.0, and the Exposure Assessment is presented in Section 4.0. Section 5.0 presents the Risk Characterization and Summary and Conclusions. Uncertainties associated with this risk assessment are discussed in Section 6.0.



2.0 HAZARD IDENTIFICATION

2.1 Constituent of Potential Concern Selection Process

The constituents to be quantitatively assessed in the risk assessment are selected in the Hazard Identification step. The constituents expected to be emitted from the Coleson Cove Generating Station depend upon the type of fuel used at the facility. Constituents of potential concern (CPCs) were identified for each set of operating conditions based upon measurements of constituents in fly ash from emissions from the CCGS, measurements of constituents in fly ash from emissions from similar facilities, measurements of constituents in fly ash from emissions from a facility using Orimulsion® as fuel, U.S. EPA sources, and other literature sources. Each constituent expected to be emitted from the facility was evaluated for its potential to pose a threat to human health. The factors taken into consideration in this process are the maximum amount of the constituent expected to be released, its toxicity, and its mobility and persistence in the environment. A subset of constituents was then selected for inclusion in the quantitative risk assessment. An average emission rate was then estimated for each of these constituents based on fly ash measurements, facility emission measurements, engineering calculations, air pollution control equipment specifications, U.S. EPA AP-42 default emission factor tables, and/or information gathered from the scientific literature.

The list of constituents selected as CPCs for each set of operating conditions is presented in Table 2-1. As shown, a total of 31 constituents were selected as CPCs in the risk assessment of the Existing Operation - Fuel Oil #6 - High Load Scenario and the Refurbishment Project - Orimulsion® - High Load Scenario. These included the following:

- Eight metals (arsenic, beryllium, cadmium, chromium (evaluated as total chromium and chromium VI, lead, mercury (evaluated as elemental mercury, methylmercury, and divalent mercury), nickel, and vanadium);
- 16 polynuclear aromatic hydrocarbons (PAH) (acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(ghi)perylene, chrysene, dibenz(ah)anthracene, fluoranthene, fluorene, indeno(123cd)pyrene, naphthalene, phenanthrene, and pyrene);
- four volatile organic compounds (VOCs) (benzene, 1,4-dichlorobenzene, formaldehyde, and toluene).

A total of 23 constituents were selected as CPCs in the risk assessment of the Natural Gas - High Load Scenario. These included the following:

- Mercury (evaluated as elemental mercury, methylmercury, and divalent mercury);
- 16 polynuclear aromatic hydrocarbons (PAH) (acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene,

benzo(ghi)perylene, chrysene, dibenz(ah)anthracene, fluoranthene, fluorene, indeno(123cd)pyrene, naphthalene, phenanthrene, and pyrene);

- four volatile organic compounds (VOCs) (benzene, 1,4-dichlorobenzene, formaldehyde, and toluene).

2.2 Emissions Estimation

Estimated emission rates for each CPC under each set of conditions are shown in Table 2-2. The emission rates shown in Table 2-2 are representative of reasonable worst case emissions. As shown below, these rates were based on the facility operating continuously at 100% capacity (24 hours per day, 365 days per year, for 30 years). Because the facility will not in actuality operate at full load continuously, actual emissions may be lower. Accordingly, the actual risks posed by the proposed facility due to these constituents are likely to be lower than the estimated risks presented in this risk assessment.

An average emission rate was then estimated for each of these compounds based on fly ash measurements, facility emission measurements, engineering calculations, air pollution control equipment specifications, U.S. EPA AP-42 default emission factor tables, and/or information gathered from the scientific literature.

Table 2-3 presents the facility and stack parameters used to generate the emission factors for each set of operating conditions. An emission factor for mercury in the Refurbishment Project - Orimulsion® - High Load Scenario (7.1×10^{-6} g/sec) was derived from vendor information and estimates of removal efficiency of mercury. This emission factor was increased by a factor of ten (from 7.1×10^{-6} g/sec to 7.1×10^{-5} g/sec) in the risk assessment as a conservative measure.

2.3 Air Quality and Deposition Modeling

Air dispersion and deposition modeling has been performed in this risk assessment to quantify the level of compounds in ambient air and other environmental media to which potential receptors may be exposed as a result of emissions from the CCGS under each of three sets of operating conditions. Compound concentrations in ambient air, predicted using air quality dispersion models, are used to evaluate potential human exposures via inhalation. Deposition algorithms estimate the magnitude of compound deposition onto soil, water body, and vegetation surfaces. This information can then be used to evaluate potential human exposures via direct and indirect pathways. This section describes the methodologies used in the dispersion and deposition modeling for the three sets of operating conditions, identifies and discusses the source input parameters used in the modeling for each set of operating conditions, and presents the results of the modeling analysis. Jacques Whitford Environment Limited of Fredericton, New Brunswick performed the air quality and deposition modeling (JWEL, 2002).

2.3.1 Air Quality Modeling Approach

An evaluation of the model selection criteria indicates that the model(s) used in this application must potentially be able to simulate point sources in a rural or urban environment and consider simple terrain receptors. The appropriate U.S. EPA guideline model for this application is the

Industrial Source Complex (ISC) model. The most recent versions of the ISC short-term (ISC-PRIME) model has been used to evaluate annual average air quality impacts for facility emissions of specific metals and organic compounds for each set of operating conditions.

The analyses presented in this report are based upon the more precise long-term average constituent concentrations predicted by a 5-year ISC-PRIME modeling analysis. These 5-year average concentrations for each receptor location have been developed from hour-by-hour ISC-PRIME modeling results as a way to represent long term average ("lifetime") concentrations. Based upon these results, a spatial distribution of the 5-year average ground level concentrations and deposition fluxes is generated. These are ultimately combined with appropriate emission rate and dose response information to evaluate chronic effects.

The ISC-PRIME dispersion model is applied in accordance with the recommendations made by U.S. EPA (1999a) and in the Guideline on Air Quality Models (U.S. EPA, 1986b, with Supplements A, B, and C), including options for air concentrations, dry deposition, wet deposition, and plume depletion.

Wet deposition consists of wet removal processes that deposit particulate and condensable pollutants in a plume onto the Earth's surface. The wet removal processes are governed by precipitation (rain or snow) which captures particulate matter and condensable pollutants as they fall through the plume. This capture process is referred to as scavenging, and is highly dependent on the particulate matter size. Larger particles are more easily scavenged by precipitation from the plume than smaller particles. To model wet deposition, the scavenging coefficient (which is dependent on particle size) is required for both rain and snow precipitation. In ISCST3 and ISC-PRIME, the wet deposition flux (which occurs only during precipitation) is calculated by multiplying a scavenging ratio (the product of a scavenging coefficient and the precipitation rate) by the vertically integrated concentration. The precipitation is assumed to originate from above the plume, so the wet deposition flux is calculated even if the plume height exceeds the mixing height.

Dry deposition is based upon the terminal velocity of the particles in the plume. Thus, it is mainly a function of the particle mass and the acceleration due to gravity. The larger particles will fall more rapidly than the smaller particles and will hence be deposited closer to the stack. To model dry deposition, the key parameters required by the model are the particle size distribution and the particle density. In ISCST3 and ISC-PRIME, the dry deposition flux is calculated as the product of the concentration and the deposition velocity. The deposition velocity is calculated using a resistance method which incorporates the effects of Brownian motion, inertial impaction, and gravitational settling. In addition to these resistances, the deposition algorithm incorporates adjustments for possible phoretic effects, including thermophoresis (particles close to a hot surface are directed away from the surface), diffusiophoresis (particles close to an evaporating surface are more likely to be impacted by a water molecule on the side of the particle facing the surface), and electrophoresis (attractive electrical forces assist the transport of small particles). These phoretic effect corrections are typically small, but can be significant for smaller particles ($0.1 - 1.0 \mu\text{m}$) with low deposition velocities (US EPA, 1995). Hence the characterization of land use over the modelled area plays an important role in the deposition predictions.

To accurately characterise the deposition of the compounds of potential concern (CPC), the deposition rates must be predicted for the three general phases in which the CPCs can exist; vapour phase, particle phase, and particle bound. The appropriate phase which is used to predict the deposition flux of each CPC is based upon physical parameters including the volatility and condensation onto existing particulate matter in the plume. Depending of the actual CPC, it is possible that fractions of the deposition may need to be partitioned among two or all three of the phases.

Highly volatile compounds, including organic compounds and mercury, do not have a dry deposition flux due to the assumption that their density is close to that of the ambient air. For vapour phase CPCs, the scavenging coefficient is dependent upon the properties of the CPC. In the absence of sufficient data to develop these coefficients, the wet deposition flux of the vapour phase CPCs are modelled using the scavenging coefficient for the smallest particle size. The deposition of these small particles is assumed to be more influenced by molecular processes than physical ones, and would hence be representative of the processes involved in vapour phase wet deposition (US EPA, 1998a).

The deposition of inorganic compounds and relatively non-volatile CPCs is modelled as particle phase deposition. For particle phase CPCs, all of the mass emitted from the plume source is assumed to exist in the particulate phase. Wet and dry deposition fluxes for particle phase CPCs are predicted according to the methods outlined above.

For organic compounds and mercury, some of the vapour will condense onto the surface of the particulate matter during transport thorough the atmosphere (referred to as particle bound), and will then settle to the Earth's surface via the wet or dry deposition of the particulate matter to which it is bound. Since this condensation is primarily controlled by mass transfer processes, the particle size distribution used in the model is based upon particle surface area (one of the rate controlling factors in mass transfer) instead of upon particle mass. The surface area weighting of particles will approximate the situation were a semivolatile organic contaminant condenses to the surface of a particle after it cools versus a mass based particle weighting which approximates the situation where the CPC originates in the stack in particle form. The wet and dry deposition fluxes for particle bound CPCs are predicted according to the methods outlined above.

For each set of operating conditions, separate modeling runs were conducted for:

- Undepleted air concentrations;
- Wet and dry deposition of particles based on mass-weighted particle distribution, including depleted air concentrations;
- Wet and dry deposition of particles based on area-weighted particle size distribution, including depleted air concentrations; and
- Wet deposition of vaporous gases.

Dispersion Environment

For the current modeling application, the ISC-PRIME model was executed for a rural environment in the regulatory mode, with the exception of missing data. ISC-PRIME was set to skip missing hours and treat that hour the same as a calm (i.e., set concentration to zero). The regulatory default mode would stop all dispersion calculations after encountering a missing data hour.

The ISC-PRIME rural dispersion option was selected for all three sets of operating conditions because the model domain within a 3 km radius of the source is predominately a rural environment with coastal terrain features (Auer, 1978). The selection of a rural environment sets ISC-PRIME to use the pre-defined temperatures and wind speed profiles for a rural area as well as rural dispersion coefficients (i.e., Pasquill-Gifford).

Terrain Characteristics

Terrain elevations were obtained from the New Brunswick Geographic Information Centre. Ground elevations for each receptor were taken from this topographical map electronically and used in the input file. The elevations determined electronically were compared with the hard copy Reference Map and there was good agreement between the two.

Terrain surrounding the generating station is relatively flat and predominantly rural, with a few zones of high relief within the 30 km distance of the source. These zones include Crane Mountain, Nowland Hill and Charlton Bluff, all located approximately 11 km to the north of the station.

The highest terrain on the grid is approximately 190 m above mean sea level (msl). This area is located on the northwest corner of the grid. There is one node at an elevation of 215 m. The stack is 197 m above msl. Thus, the stack height is greater than the terrain height over essentially the complete grid, and the terrain category from a modeling point of view is considered to be "simple terrain."

Point Source Characteristics

The source data required to run the model includes the following parameters for each emission point: the physical location of the stack or vent, the emission rate, the release height above grade, the exhaust gas exit velocity, the inside diameter of the stack or vent, and the exhaust gas temperature at the release point. Optional inputs include source elevation, building heights and widths, particle sizes with settling data, and surface reflection coefficients.

The exiting stack (in the Existing Operation - Fuel Oil #6 - High Load Scenario and the Natural Gas - High Load Scenario) and the proposed new stack (in the Refurbishment Project - Orimulsion® - High Load Scenario) are considered tall stacks in the modeling sense (according to the "Guideline for Determination of Good Engineering Practice Stack Height," (U.S. EPA, 1985), and therefore, building wake effects are not expected to be significant. Nevertheless, the

enhanced downwash algorithms contained in the ISC-PRIME model were used to assess the potential for downwash effects to occur for all sets of operating conditions.

The site plan (see Figure 2-1) shows the location of the generating station and the existing 183 m high stack with three flues, one for each 350 MW boiler and turbine unit. The proposed location of the new FGD stack (applicable only to the Refurbishment Project - Orimulsion® - High Load Scenario) is also presented in Figure 2-1. The proposed design of the new stack includes two flues, each expected to handle exhaust flows associated with approximately 525 MW of power generation, for a total of 1050 MW, at full load.

For all three sets of operating conditions, the volumetric flow rates of exhaust gas from each of the flues were combined by assuming the total exhaust flows through a single cross-sectional area equivalent to the area of the flues in the stack. Thus, the total flow from the stack was modeled as being emitted from one release point with an equivalent stack diameter. This is a realistic approach, since under this modeling assumption, the benefit from the momentum and buoyancy fluxes of the combined flues are realized in the analysis.

The load conditions with their respective stack parameters used to model the three sets of operating conditions are provided in Tables 2-3.

Meteorological Data

The weather data required to conduct the dispersion modelling (using ISC-PRIME) includes wind velocities and direction, temperature, atmospheric stability, and mixing layer depth on an hourly basis. Wind and temperature data are readily available from local meteorological stations. Atmospheric stability and mixing layer depths are calculated from additional raw meteorological data including cloud cover, snow cover and solar radiation.

Based on consultation with the local Atmospheric Environment Service climatologist, the most appropriate weather station to be used to dispersion modeling purposes is the Saint John Airport station for surface data, and the Yarmouth, NS station for upper air data (Richards, 2000).

Quality assured raw hourly meteorological data from the beginning of 1996 to the end of December 2000 were obtained from the Atmospheric Environment Service for the Saint John Airport. Twice daily, upper air sounding data were obtained for the Yarmouth, NS weather station, the nearest upper air station to Saint John. The raw data for the area were used to calculate stability parameters and mixing layer depths (mixing heights) with the aid of the U.S. EPA PCRAMMET meteorological pre-processor (U.S. EPA, 1999a). PCRAMMET was used to merge the surface data set with the upper air data, to provide a quality checked meteorological data set. The PCRAMMET processor requires hourly estimates of wind speed, direction, temperature, cloud cover as well as the 1200 GMT (7:00 am SLT) sounding to generate the requisite data for modeling.

The annual average joint frequency distribution of wind speed and direction for the Saint John area is presented in Figure 2-2. The wind direction is reported as the direction from which the

wind blows, and is measured at a height of 10 m above grade. The dominant winds are from the south and southwesterly quadrants, with the northwesterly direction also being important, though not as dominant. Very low wind speeds occur most frequently from the east quadrant. Strong winds occur most often from the southwest sector.

The stability of the atmosphere refers to the amount of vertical motion in the atmosphere, and this reflects the ability of the atmosphere to disperse and dilute constituents in air. As might be expected, constituents in air being released into a wind field in a stable atmosphere would experience less vertical motion than if dispersed in an unstable atmosphere, and therefore there would be less mixing and less dilution as the plume moves downwind. The atmosphere is termed unstable when there is good vertical mixing, for example on a warm sunny day with light winds. A stable atmosphere results when the surface of the earth is cooler than the air above it, for example on a clear, cool night. The stable atmosphere generally exists with a temperature inversion at a particular height above grade. The atmospheric stability is considered neutral when there is little or no heating or cooling of the ground and the air adjacent to it. This neutral stability is characterized by cloudy conditions which inhibit solar radiation, windy conditions which would cause rapid vertical mixing, or a transition near sunrise or sunset when the atmosphere is changing from unstable to stable or vice versa.

For convenience in the prediction of plume dispersion, different schemes have been developed to classify different levels of stability. The historic Pasquill-Gifford scheme has been widely used, that consists of six categories A through F, where A is highly unstable (lots of vertical motion), Class D is neutral and Class F is very stable, and modified by Turner (1994). Based on consultation with the Environment Canada personnel (Beattie, 2002), the stabilities for June, July and August were revised to better represent the actual stabilities during those seasons, for times when the wind blows from the water toward land.

The distribution of atmospheric stability from 1995-2000 at the Saint John Airport is presented in Table 2-4. The neutral or near neutral Classes (C and D) occur about 66% of the time. The stable conditions occur about 16% of the time.

The revised distribution of atmospheric stability from 1995-2000 at the Saint John airport is also presented in Table 2-4. As shown, the neutral or near neutral Classes (C and D) are the same, as expected. There is a slight decrease in Classes A and B, and a corresponding increase in Classes E and F.

Receptor Grid

For each set of operating conditions, ambient concentrations were predicted at locations in the area surrounding the generating station. For the purposes of modeling, a Cartesian receptor grid was established consisting of grid nodes spaced 100 m apart extending from the centroid of the source out to 3 kilometers from the centroid. The location of the centroid is the center of the existing stack. For distances from three kilometers to ten kilometers, the node spacing was increased to 1000 meters and for distances of 10 kilometers to 20 kilometers, the node spacing was 2000 meters. Additional receptor nodes were added in the area of the City of Saint John to

ensure that all areas were sufficiently "covered" by receptor nodes. Other "sensitive" locations (e.g., the nearest farm or water body) of importance to multipathway risk assessment could be selected from the guided locations. There are an adequate number of receptor locations so that representative or slightly conservative air concentrations or deposition fluxes could always be identified. Figure 2-3 shows the receptor grid used to model all three sets of operating conditions.

2.3.2 Particle-Size Distributions

Due to insufficient particle size distribution data available for facilities using Orimulsion® fuel and similar pollution control equipment, the generalized particle size distribution presented in *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities* (US EPA, 1998a) was used. This distribution, presented in Table 2-5, has been designated by the USEPA to be used as the default distribution if site-specific data is unavailable, and is applicable for combustion facilities equipped with either ESPs or fabric filters.

Two types of classification data used for modeling analysis, distribution by particle mass, and by particle surface areas are presented in Table 2-5. As discussed in Section 2.3.3, this accounts for compounds which may be distributed by particle mass while others may be distributed on the surface of particles.

2.3.3 Surface Deposition Analysis

The evaluation of exposure through pathways other than inhalation requires an analysis of surface deposition. Dry deposition of particulates occurs when emissions are transported downward through gravitational settling and turbulence and attach to surfaces. Wet deposition can occur when particulates in the plume are "washed out" by falling precipitation.

Dry Deposition

Dry deposition is a process by which both particulate and gaseous species may be removed from the air above a surface. The relationship between the concentration in the air and that in the surface material requires movement of material from the one medium to the other. This movement can be described as a rate of mass transfer per unit area of surface (i.e., a flux with units of mass ÷ area x time, such as g/m²-yr).

The ratio of this mass transfer rate to the difference in concentration between the two media can be thought of as a velocity of transfer since it has velocity units (e.g., cm/sec or m/yr). The exchange of mass is related to the microscale characteristics of the surface and the atmospheric turbulence, and thus the most successful models for this process have been those which have struck a balance between the need for addressing the rather complex details of the surface processes, and the need to use relatively simple analytical models that have been validated by comparison with the limited measurement data sets that exist. In its simplest form, the deposition velocity, V_D , is defined practically as the ratio of the flux, F_D , of material of interest to its air concentration, χ_A :

$$V_D \approx F_D/\chi_A$$

This definition assumes that the concentration at the surface available for re-emission to the air is negligible. The ISC-PRIME dry deposition model is based on a dry deposition algorithm developed for the Acid Deposition and Oxidant Model (ADOM) (U.S. EPA, 1986a). The general approach used in this algorithm is to include explicit parameterizations of the effects of Brownian motion, inertial impaction, and gravitational settling. The deposition velocity is also a function of turbulence defined by factors such as wind speed, stability, and surface roughness as well as particle size and density. Stability, wind speed, and other parameters are provided by the meteorological input file used for this portion of the risk assessment.

Dry deposition fluxes are computed from the product of two factors, the ground-level concentration and a deposition velocity (V_D). In modeling dry deposition, ISC-PRIME also accounts for plume depletion of airborne CPCs near the surface due to particulate settling and computes dry deposition of vapors. The algorithms employed are based on those used in the CALPUFF model and operate on the same general principal as that described above.

Wet Deposition

A scavenging ratio approach is used to model the deposition of gases and particles through wet removal in ISC-PRIME. The model is similar to the algorithm previously adopted by Bowman et al. (1987) and U.S. EPA for COMPDEP. The model uses hourly rainfall amounts to calculate the rain rate that is used to estimate the scavenging coefficient for each hour. The model requires hourly precipitation, wind variables, and the size and fractional distribution of emitted particles. As recommended by the US EPA (1998a), the scavenging coefficients were based upon the relationship developed by Jindal and Heinhold in 1991 between scavenging coefficients and particle size. It has been indicated in research that for some pollutants, the frozen precipitation scavenging coefficients are approximately 1/3 of the liquid precipitation scavenging coefficients. Since the research data is somewhat limited with respect to the types of pollutants studied, a conservative approach was taken by assuming that the frozen precipitation scavenging coefficients were equal to the liquid ones. Table 2-6 shows the scavenging coefficients used in deposition modeling.

2.3.4 Transport and Deposition of Mercury

Much research recently has focused upon the special deposition characteristics of mercury emissions from incinerator (as well as power plant) stacks. There are many remaining questions concerning the behavior of mercury in the atmosphere and at the point of deposition and uptake into plants or soil. U.S. EPA (1998a) recognizes that stack emissions contain mercury in both vapor and particulate form. Further, vapor emissions are believed to include both elemental (Hg^0) and oxidized (e.g., Hg^{2+}) species, while particulate emissions are believed to be primarily oxidized species. The speciation of mercury likely depends upon the fuel source, operating conditions, and emission controls.

The majority of mercury emissions are in the vapor form, although some particle-bound mercury is believed to be emitted. All mercury emitted is believed to be emitted either as Hg^0 or Hg^{2+} . Among the total mercury emissions, a majority of total emissions of mercury are not deposited and are considered to be vertically diffused to the free atmosphere to become part of the global cycle (U.S. EPA, 1998a). U.S. EPA (1998a) recommends assuming that 48% of total emitted mercury is emitted as Hg^{2+} and 0.2% of total emitted mercury is emitted as Hg^0 . These fractions account for the majority of total emitted mercury that is lost to the global cycle. Methylmercury is not assumed to be emitted from the stack.

The fraction of total mercury emissions that is assumed to be emitted as Hg^{2+} (48%) is evaluated via both inhalation and indirect exposure (deposition onto surfaces and uptake into plants and animal products). The fraction of total mercury emissions that is assumed to be emitted as vapor Hg^0 is evaluated via inhalation only.

U.S. EPA (1998a) recommends assuming that a fraction of the Hg^{2+} that deposits on surfaces may become methylated. In soil, 98% of deposited Hg^{2+} remains as Hg^{2+} , while the remaining 2% of deposited Hg^{2+} becomes methylmercury (U.S. EPA, 1998a). Among the total mercury that deposits on water bodies, 85% is assumed to remain Hg^{2+} , and 15% is assumed to be methylmercury. Mercury can also enter water bodies via runoff and erosion. U.S. EPA (1998a) recommends that 2% of the mercury in this watershed soil is methylmercury and enters the water body as such. Additionally, U.S. EPA (1998a) 15% of the Hg^{2+} in watershed soil that enters the water body as a result of runoff and erosion becomes methylated in the water body. In recommending that 15% of Hg^{2+} entering the waterbody becomes methylated, U.S. EPA (1998a) refers to the Mercury Study Report to Congress (U.S. EPA, 1997b), as a source for the "typical" amount of methylmercury, as a proportion of total mercury concentration in various media. The 2% methylmercury content of soils is consistent with that reported in U.S. EPA (1997b). However, the 15% default for the fraction of the mercury concentration in surface water that becomes methylated is not supported by the reference cited in U.S. EPA (1998a). U.S. EPA (1997b) derives proportional methylmercury content of surface water bodies from reports on individual water bodies and (on page 3-10 of Volume III of the Report) states: "*levels around 10% being the most common.*"

This section of U.S. EPA (1997b) contains a Table (Table 3-7) containing a summary of five original studies reporting methylmercury as a fraction of total mercury concentration. No percentage listed on the Table exceeds 14%. Appendix B to Volume III of U.S. EPA (1997b), which contains the methodology for derivation of the BAFs cited in the draft U.S. EPA (1998a), provides a statistical analysis of the proportional water concentration of methylmercury in the epilimnion (the epilimnion is that portion of the water column above a point of temperature change, or thermocline) and report on page D-22 that the most appropriate point estimate from the data collected is 7.8%.

Because U.S. EPA (1998a) has misused the data and conclusions of the Mercury Study Report to Congress (U.S. EPA, 1997b), the risk assessment will use a value of 7.8% as the appropriate value for proportional methylmercury concentration for freshwater.

2.4 Air Modeling Results

2.4.1 Normalized Air Concentrations

For each of the modeled sets of operating conditions, the 5-year average normalized ambient air concentrations (in $\mu\text{g}/\text{m}^3$ for a source strength of 1 g/sec) calculated by the ISC-PRIME model are presented in figures as a graphical overlay on the map of the 30 km radius study area. Also indicated on the base map for this graph are the specific receptor locations which are described in detail in Section 4.0. These normalized concentrations are presented for these specific receptor locations in Table 2-7 for the Existing Operation - Fuel Oil #6 - High Load Scenario, Table 2-8 for the Refurbishment Project - Orimulsion® - High Load Scenario, and Table 2-9 for Natural Gas - High Load Scenario. Appendix A contains the complete output from dispersion and deposition modeling for all receptor nodes for each set of operating conditions.

Figure 2-4 shows the normalized air concentrations of compounds emitted in particle-bound form for the Existing Operation - Fuel Oil #6 - High Load Scenario and Natural Gas - High Load Scenario. Normalized concentrations of compounds emitted in particle-phase form in the Existing Operation - Fuel Oil #6 - High Load Scenario and Natural Gas - High Load Scenario are shown on Figure 2-5. Figure 2-6 shows the normalized air concentrations of compounds emitted in vapor form for Existing Operation - Fuel Oil #6 - High Load Scenario and Natural Gas - High Load Scenario.

Figure 2-7 shows the normalized air concentrations of compounds emitted in particle-bound form for Refurbishment Project - Orimulsion® - High Load Scenario. Normalized concentrations of compounds emitted in particle-phase form in the Refurbishment Project - Orimulsion® - High Load Scenario are shown on Figure 2-8. Figure 2-9 shows the normalized air concentrations of compounds emitted in vapor form for Refurbishment Project - Orimulsion® - High Load Scenario.

2.4.2 Deposition Fluxes

The comparable normalized deposition fluxes are presented for the two size distributions utilized to represent the different types of emitted constituents: those that adhere to the surface (surface-weighted), and those which make up the volume (mass weighted) of the depositing particles.

These normalized dry deposition rates are presented for these specific receptor locations in Table 2-7 for the Existing Operation - Fuel Oil #6 - High Load Scenario, Table 2-8 for the Refurbishment Project - Orimulsion® - High Load Scenario, and Table 2-9 for Natural Gas - High Load Scenario.

Figure 2-10 shows the normalized total deposition rates of compounds emitted in particle-bound form for Existing Operation - Fuel Oil #6 - High Load Scenario and Natural Gas - High Load Scenario. Normalized total deposition rates of compounds emitted in particle-phase form in the Existing Operation - Fuel Oil #6 - High Load Scenario and Natural Gas - High Load Scenario are shown on Figure 2-11. Figure 2-12 shows the normalized wet deposition rates of compounds emitted in vapor form for Existing Operation - Fuel Oil #6 - High Load Scenario and Natural Gas - High Load Scenario.

Figure 2-13 shows the normalized total deposition rates (dry + wet) of compounds emitted in particle-bound form for Refurbishment Project - Orimulsion® - High Load Scenario. Normalized total deposition rates of compounds emitted in particle-phase form in the Refurbishment Project - Orimulsion® - High Load Scenario are shown on Figure 2-14. Figure 2-15 shows the normalized wet deposition rates of compounds emitted in vapor form for Refurbishment Project - Orimulsion® - High Load Scenario.

Table 2-1
CPCs Evaluated in the Risk Assessment
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Existing Operation - Fuel Oil #6 - High Load Scenario	Refurbishment Project - Orimulsion - High Load Scenario	Natural Gas - High Load Scenario
Mercury, elemental Mercury, methyl Mercury, divalent 1,4-Dichlorobenzene Acenaphthene Acenaphthylene Anthracene Arsenic Benzene Benzo(a)anthracene Benzo(a)pyrene Benzo(b)fluoranthene Benzo(k)fluoranthene Benzo(g,h,i)perylene Beryllium Cadmium Chromium Chromium VI Chrysene Dibenzo(a,h)anthracene Fluoranthene Fluorene Formaldehyde Indeno(1,2,3-cd)pyrene Lead Naphthalene Nickel Phenanthrene Pyrene Toluene Vanadium	Mercury, elemental Mercury, methyl Mercury, divalent 1,4-Dichlorobenzene Acenaphthene Acenaphthylene Anthracene Arsenic Benzene Benzo(a)anthracene Benzo(a)pyrene Benzo(b)fluoranthene Benzo(k)fluoranthene Benzo(g,h,i)perylene Beryllium Cadmium Chromium Chromium VI Chrysene Dibenzo(a,h)anthracene Fluoranthene Fluorene Formaldehyde Indeno(1,2,3-cd)pyrene Lead Naphthalene Nickel Phenanthrene Pyrene Toluene Vanadium	Mercury, elemental Mercury, methyl Mercury, divalent 1,4-Dichlorobenzene Acenaphthene Acenaphthylene Anthracene Benzene Benzo(a)anthracene Benzo(a)pyrene Benzo(b)fluoranthene Benzo(k)fluoranthene Benzo(g,h,i)perylene Chrysene Dibenzo(a,h)anthracene Fluoranthene Fluorene Formaldehyde Indeno(1,2,3-cd)pyrene Naphthalene Phenanthrene Pyrene Toluene

Table 2-2
Emission Factors
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

	Existing Operation - Fuel Oil #6 - High Load Scenario	Refurbishment Project - Orimulsion - High Load Scenario	Natural Gas - High Load Scenario
Chemical	Value (g/s)	Value (g/s)	Value (g/s)
Mercury, elemental	0.000483	0.000071	0.00000134
1,4-Dichlorobenzene	0.000549	0.000181	0.00159
Acenaphthene	0.000162	0.0000496	0.00000238
Acenaphthylene	0.00000194	0.000000595	0.00000238
Anthracene	0.00000936	0.00000287	0.00000317
Arsenic	0.00121	0.0000873	NA
Benzene	0.00182	0.00213	0.00277
Benzo(a)anthracene	0.0000308	0.00000943	0.00000238
Benzo(a)pyrene	0.00000845	0.00000279	0.00000159
Benzo(b)fluoranthene	0.0000235	0.00000776	0.00000238
Benzo(k)fluoranthene	0.0000235	0.00000776	0.00000238
Benzo(g,h,i)perylene	0.0000173	0.00000532	0.00000159
Beryllium	0.000044	0.00000502	NA
Cadmium	0.000033	0.0000024	NA
Chromium	0.00198	0.000207	NA
Chromium VI	0.000574	0.0000601	NA
Chrysene	0.0000183	0.0000056	0.00000238
Dibenzo(a,h)anthracene	0.0000128	0.00000393	0.00000159
Fluoranthene	0.0000371	0.0000114	0.00000396
Fluorene	0.0000343	0.0000105	0.0000037
Formaldehyde	0.281	0.00607	0.0991
Indeno(1,2,3-cd)pyrene	0.0000164	0.00000504	0.00000238
Lead	0.0195	0.00000742	NA
Napthalene	0.00867	0.000905	0.000806
Nickel	0.572	0.079	NA
Phenanthrene	0.0000805	0.0000247	0.0000225
Pyrene	0.0000326	0.00001	0.00000661
Toluene	0.0528	0.0271	0.00449
Vanadium	3.76	0.343	NA

Sources:

"Locating and Estimating Air Emissions from Sources of Hg and Hg Compounds", USEPA Dec 97

US EPA, AP-42, 9/98

Dec. 1998 flyash test at Coleson Cove

"Locating and Estimating Air emissions from sources of POM", US EPA, July 1998

US EPA, AP-42, 7/98

US EPA AP-42, 2002

"Environmental Impacts of the Use of Orimulsion", USEPA July 2001

Orimulsion Data Sheets

Dec. 1998 Dalhousie flyash analysis

USEPA factor for risk assessment of electric utility industry

Golder Associates

Equipment Manufacturer Efficiency Values

NB Power emission measurements

NA - Not applicable. This compound was not assumed to be emitted for this set of operating conditions.

Table 2-3

Stack Parameters Used to Evaluate Each Operating Condition
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Parameter	Existing Operation - Fuel Oil #6 - High Load Scenario	Refurbishment Project - Orimulsion High Load Scenario	Natural Gas - High Load Scenario
Stack Height (m)	182.9	182.9	182.9
Number of flues	3	2	3
Equivalent Stack Diameter (m)	8.487	10.99	8.487
Flowrate (m ³ /s)	1697	1,428	1697
Exit Velocity (m/s)	30	15.06	30
Exit Temperature (C)	170	61	170

Source: JWEL, 2002. (Air Quality Report).

Table 2-4
 Stability Class Distribution (1995 - 2000)
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Stability Class	Percent Frequency of Occurrence	Revised Percent Frequency of Occurrence
A	0.25	0.16
B	2.7	1.9
C	8.6	8.6
D	57.2	57.2
E	8.25	9.1
F	7.7	7.8
Calm & missing data	15.3	15.3

Source:
 JWEL, 2002. (Air Quality Report).

Table 2-5
 Generalized Particle Size Distribution
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Mean Particle Diameter (μm)	Particle Radius (μm)	Surface Area/Volume (μm)	Fraction of Total Mass	Proportion of Available Surface Area	Fraction of Total Surface Area
> 15	7.50	0.400	0.128	0.0512	0.0149
12.5	6.25	0.480	0.105	0.0504	0.0146
8.1	4.05	0.741	0.104	0.0771	0.0224
5.5	2.75	1.091	0.073	0.0796	0.0231
3.6	1.80	1.667	0.103	0.1717	0.0499
2.0	1.00	3.000	0.105	0.3150	0.0915
1.1	0.55	5.455	0.082	0.4473	0.1290
0.7	0.40	7.500	0.076	0.5700	0.1656
< 0.7	0.40	7.500	0.224	1.6800	0.4880

Source: U.S. EPA, 1998a.

Table 2-6
Particle Phase and Particle Bound Scavenging Coefficients
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Mean Particle Diameter (μm)	Liquid Scavenging Coefficient	Frozen Scavenging Coefficient
> 15	6.7E-04	6.7E-04
12.5	6.7E-04	6.7E-04
8.1	5.2E-04	5.2E-04
5.5	3.9E-04	3.9E-04
3.6	2.6E-04	2.6E-04
2.0	1.3E-04	1.3E-04
1.1	6E-05	6E-05
0.7	5E-05	5E-05
< 0.7	7E-05	7E-05

Source: U.S. EPA, 1998a.

Table 2-7
Air Quality Modeling Results for Existing Operation - Fuel Oil #6 - High Load Scenario
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Cornville, New Brunswick

Unitized Air Modeling Results	Symbol	Units	Location:				
			Maximum Hypothetical Residence	Saint John	Northeast Farm	Lakes Watershed	Spruce, Ludgate, Menzies Lakes
Unitized Wet deposition - Particle bound	(Dywp_pb)	(g/m2-y) / (g/s)	0.03182	0.0006	0.00037	0.00126	0.00113
Unitized Wet deposition - Particle phase	(Dywp)	(g/m2-y) / (g/s)	0.08126	0.00098	0.00058	0.00235	0.00222
Unitized Dry deposition - Particle bound	(Dydp_pb)	(g/m2-y) / (g/s)	0.00001	0.000078	3.44E-05	0.00002	0.000013
Unitized Dry deposition - Particle phase	(Dydp)	(g/m2-y) / (g/s)	0.00001	0.00032	0.00014	0.00011	0.000064
Unitized Air concentration - Particle bound	(cyp_pb)	(ug/m3) / (g/s)	0.00001	0.00043	0.00021	0.00014	0.0000896
Unitized Air concentration - Particle phase	(cyp)	(ug/m3) / (g/s)	0.00001	0.00036	0.00018	0.00012	0.00008
Unitized Wet deposition - Vapors	(Dywpv)	(g/m2-y) / (g/s)	0.03379	0.0007	0.00043	0.00148	0.0013
Unitized Air concentration - Vapors	(Cyv)	(ug/m3) / (g/s)	0.00001	0.00045	0.00022	0.00015	0.000093
Unitized Wet+Dry deposition - Particle bound	(Dytwp_pb)	(g/m2-y) / (g/s)	0.03183	0.00067	0.00040	0.00128	0.00114
Unitized Wet+Dry deposition - Particle phase	(Dytwp)	(g/m2-y) / (g/s)	0.08128	0.0013	0.00072	0.00245	0.00222

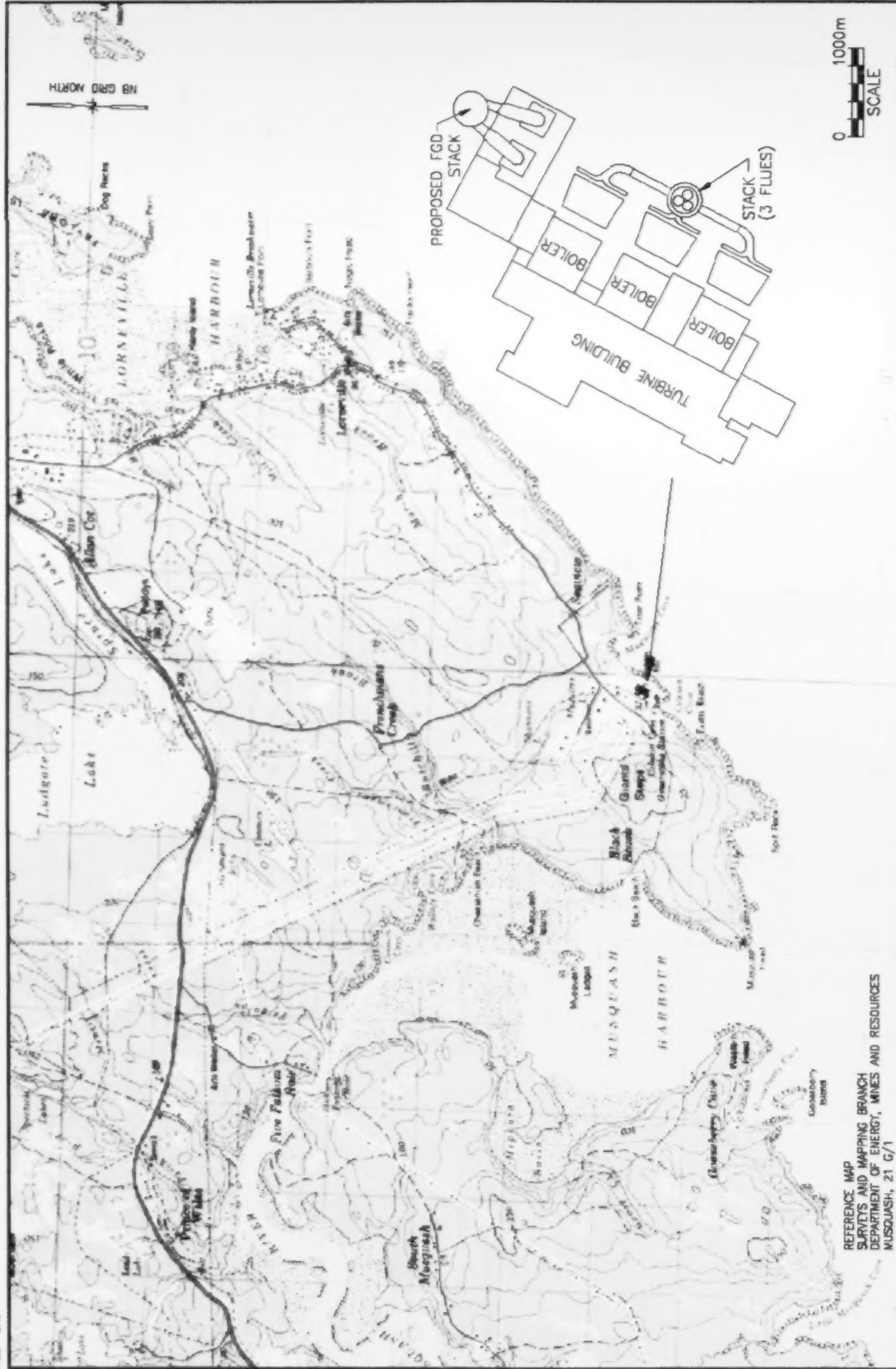
Table 2-8
Air Quality Modeling Results for Refurbishment Project - Orimulsion - High Load Scenario
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Unitized Air Modeling Results	Symbol	Units	Location:			
			Maximum Hypothetical Residence	Saint John	Northeast Farm	Lakes Watershed
Unitized Wet deposition - Particle bound	(Dywp_pb)	(g/m ² -y) / (g/s)	0.04059	0.000599	0.00037	0.00129
Unitized Wet deposition - Particle phase	(Dywp)	(g/m ² -y) / (g/s)	0.10442	0.000974	0.00057	0.00242
Unitized Dry deposition - Particle bound	(Dydp_pb)	(g/m ² -y) / (g/s)	0.00001	0.000194	7.89E-05	0.00009
Unitized Dry deposition - Particle phase	(Dydp)	(g/m ² -y) / (g/s)	0.00001	0.000803	0.00033	0.00038
Unitized Air concentration - Particle bound	(cyp_pb)	(ug/m ³) / (g/s)	0.00001	0.001160	0.00057	0.00051
Unitized Air concentration - Particle phase	(cyp)	(ug/m ³) / (g/s)	0.00001	0.000966	0.00047	0.00045
Unitized Wet deposition - Vapors	(Dyww)	(g/m ² -y) / (g/s)	0.0429	0.000701	0.00044	0.00152
Unitized Air concentration - Vapors	(Cw)	(ug/m ³) / (g/s)	0.00001	0.001209	0.00059	0.00052
Unitized Wet+Dry deposition - Particle bound	(Dytwp_pb)	(g/m ² -y) / (g/s)	0.0406	0.000794	0.00045	0.00138
Unitized Wet+Dry deposition - Particle phase	(Dytwp)	(g/m ² -y) / (g/s)	0.10443	0.001776	0.00090	0.00279
						Spruce, Ludgate, Menzies Lakes
						0.00113
						0.00217
						5.43E-05
						0.00024
						0.00033
						0.00030
						0.00130
						0.00034
						0.00118
						0.00241

Table 2-9
Air Quality Modeling Results for Natural Gas - High Load Scenario
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Unitized Air Modeling Results	Symbol	Units	Location:				
			Maximum Hypothetical Residence	Saint John	Northeast Farm	Lakes Watershed	Spruce, Ludgate, Menzies Lakes
Unitized Wet deposition - Particle bound	(Dywp_pb)	(g/m ² -y) / (g/s)	0.03182	0.0006	0.00037	0.00126	0.00113
Unitized Wet deposition - Particle phase	(Dywp)	(g/m ² -y) / (g/s)	0.08126	0.00098	0.00057667	0.00235	0.00222
Unitized Dry deposition - Particle bound	(Dydp_pb)	(g/m ² -y) / (g/s)	0.00001	0.000078	3.4444E-05	0.00002	0.000013
Unitized Dry deposition - Particle phase	(Dydp)	(g/m ² -y) / (g/s)	0.00001	0.00032	0.00014	0.00011	0.000064
Unitized Air concentration - Particle bound	(cyp_pb)	(ug/m ³) / (g/s)	0.00001	0.00043	0.00021333	0.00014	0.0000896
Unitized Air concentration - Particle phase	(cyp)	(ug/m ³) / (g/s)	0.00001	0.00036	0.00018	0.00012	0.00008
Unitized Wet deposition - Vapors	(Dywpv)	(g/m ² -y) / (g/s)	0.03379	0.0007	0.00043222	0.00148	0.0013
Unitized Air concentration - Vapors	(Cyv)	(ug/m ³) / (g/s)	0.00001	0.00045	0.0002222	0.00015	0.000093
Unitized Wet+Dry deposition - Particle bound	(Dytwp_pb)	(g/m ² -y) / (g/s)	0.03183	0.00067	0.00040333	0.00128	0.00114
Unitized Wet+Dry deposition - Particle phase	(Dytwp)	(g/m ² -y) / (g/s)	0.08128	0.0013	0.00071667	0.00245	0.00222

FILE: SITE



REFERENCE MAP
SURVEYS AND MAPPING BRANCH
DEPARTMENT OF ENERGY, MINES AND RESOURCES
MUSQUASH, 21 G/1

SITE PLAN NB POWER, COLESON COVE LORNEVILLE, NB

Date: 2001 10 29
Scale: 1 : 60 000
Job No.: 13269

Figure 2-1



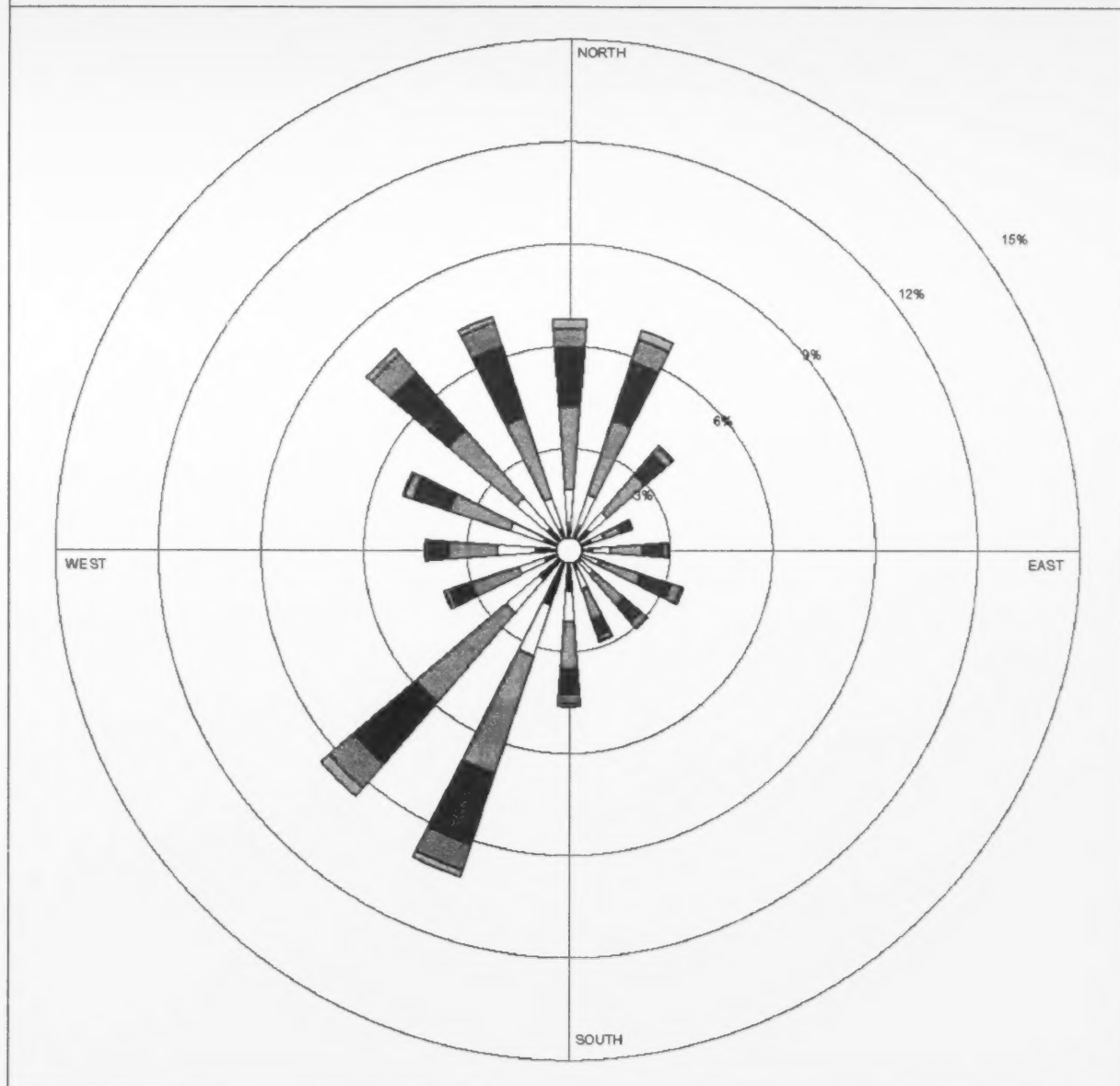
Jacques Whitford
Consulting Engineers
Environmental Scientists

THE ENGINEER IS NOT RESPONSIBLE FOR THE ACCURACY OF THE DATA OR THE RESULTS OF THE ANALYSIS OR THE DESIGN OF THE PROJECT OR THE CONSTRUCTION OF THE PROJECT.

WIND ROSE PLOT

NB Power - Coleson Cove, Lorneville, New Brunswick

Job No. 13269-0001



Wind Speed (m/s)



> 11.06
8.49 - 11.06
5.40 - 8.49
3.34 - 5.40
1.80 - 3.34
0.51 - 1.80

MODELER

JWEL

DATE

9/17/01

COMPANY NAME

Jacques Whitford Environment Ltd.

DISPLAY

Wind Speed

UNIT

m/s

COMMENTS

Meteorological Data
St. John, NB
1995-2000

AVG. WIND SPEED

4.73 m/s

CALM WINDS

15.32%

ORIENTATION

Direction
(blowing from)

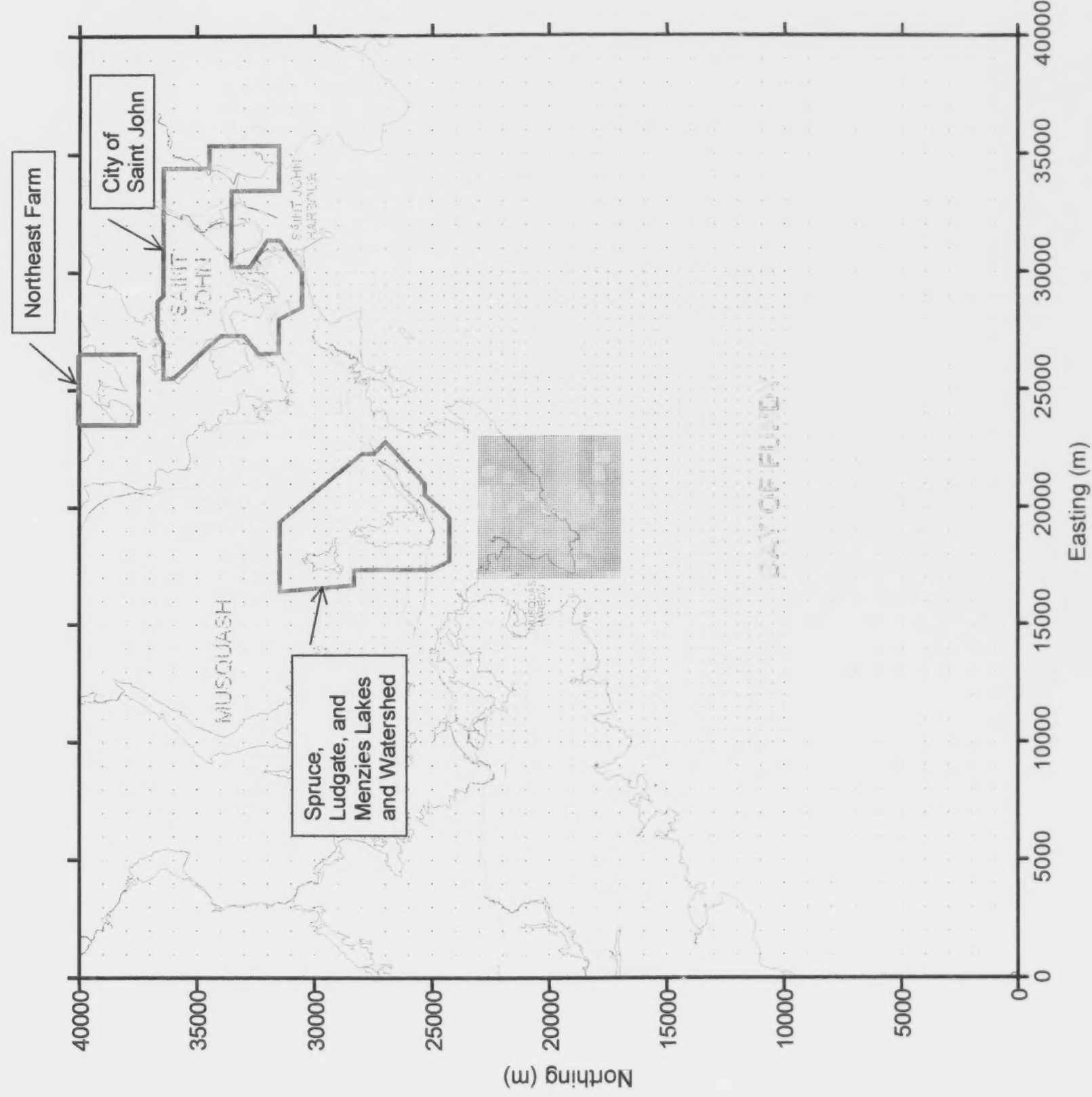
PLOT YEAR-DATE-TIME

1995
Jan 1 - Dec 31
Midnight - 11 PM

PROJECT/PLOT NO.

Figure 2-2

Figure 2-3
 Air Quality Modeling Receptor Plan
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick



LEGEND

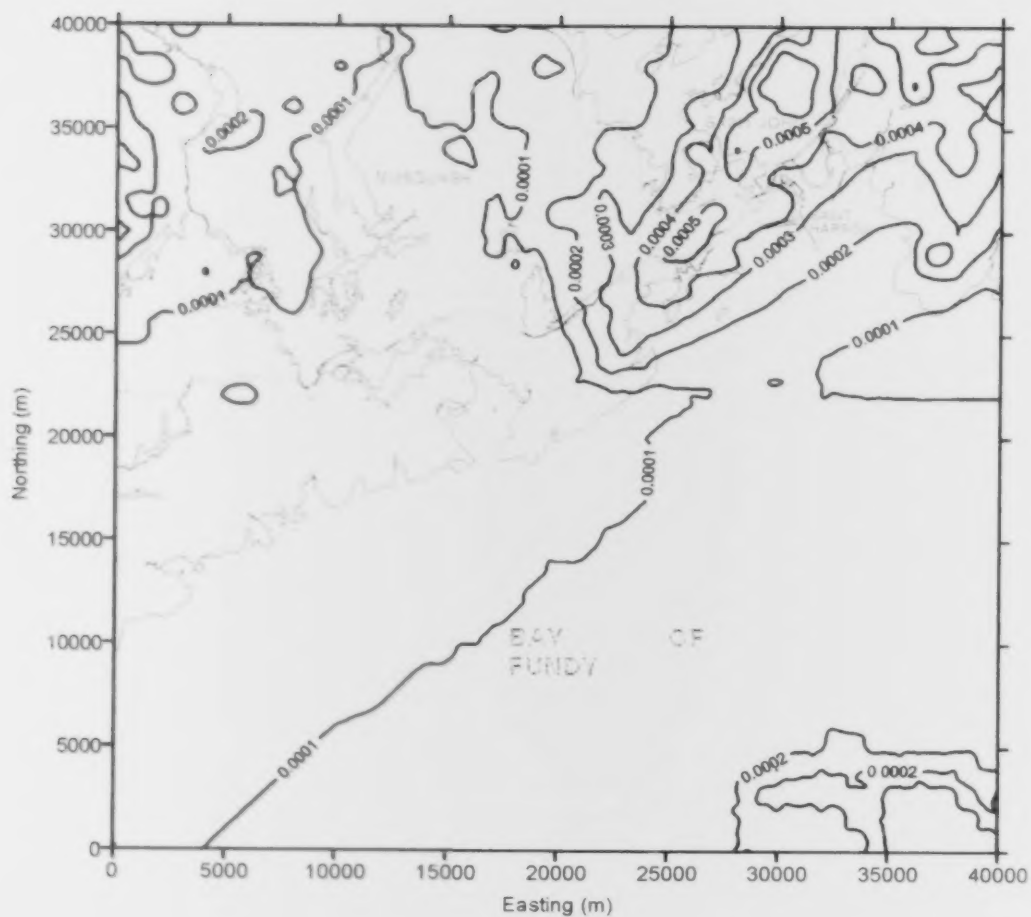
• Coleson Cove
 Generating Station

Job No. 13269-0001



Jacques Whitford
 Environment Limited

Figure 2-4
 Predicted Unitized Annual Average Air Concentrations $[(\mu\text{g}/\text{m}^3) / (\text{g}/\text{sec})]$
 Particle-Bound Form
 Existing Condition - Fuel Oil #6 - High Load Scenario
 Natural Gas - High Load Scenario
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick



LEGEND

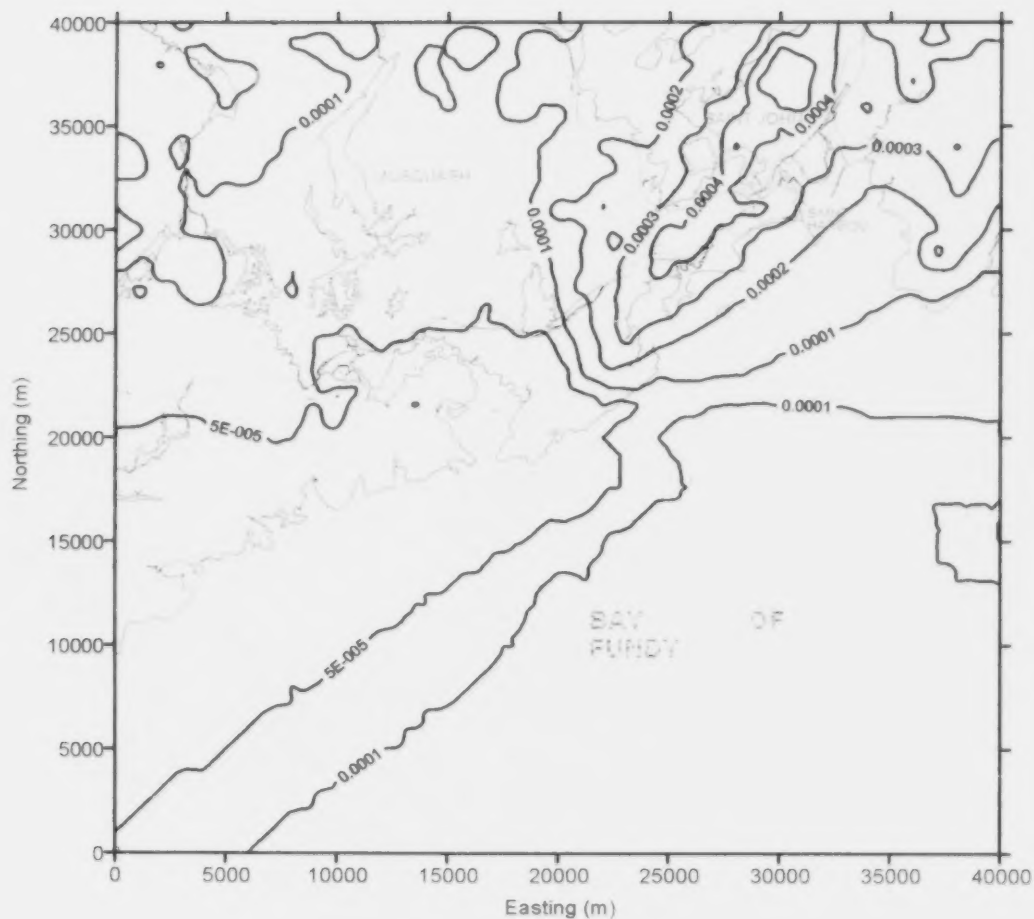
- Coleson Cove
Generating Station
- - - Concentration, $\mu\text{g}/\text{m}^3$

Job No. 13269-0001



Jacques Whitford
Environment Limited

Figure 2-5
Predicted Unitized Annual Average Air Concentrations [($\mu\text{g}/\text{m}^3$) / (g/sec)]
Particle-Phase Form
Existing Condition - Fuel Oil #6 - High Load Scenario
Natural Gas - High Load Scenario
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick



LEGEND

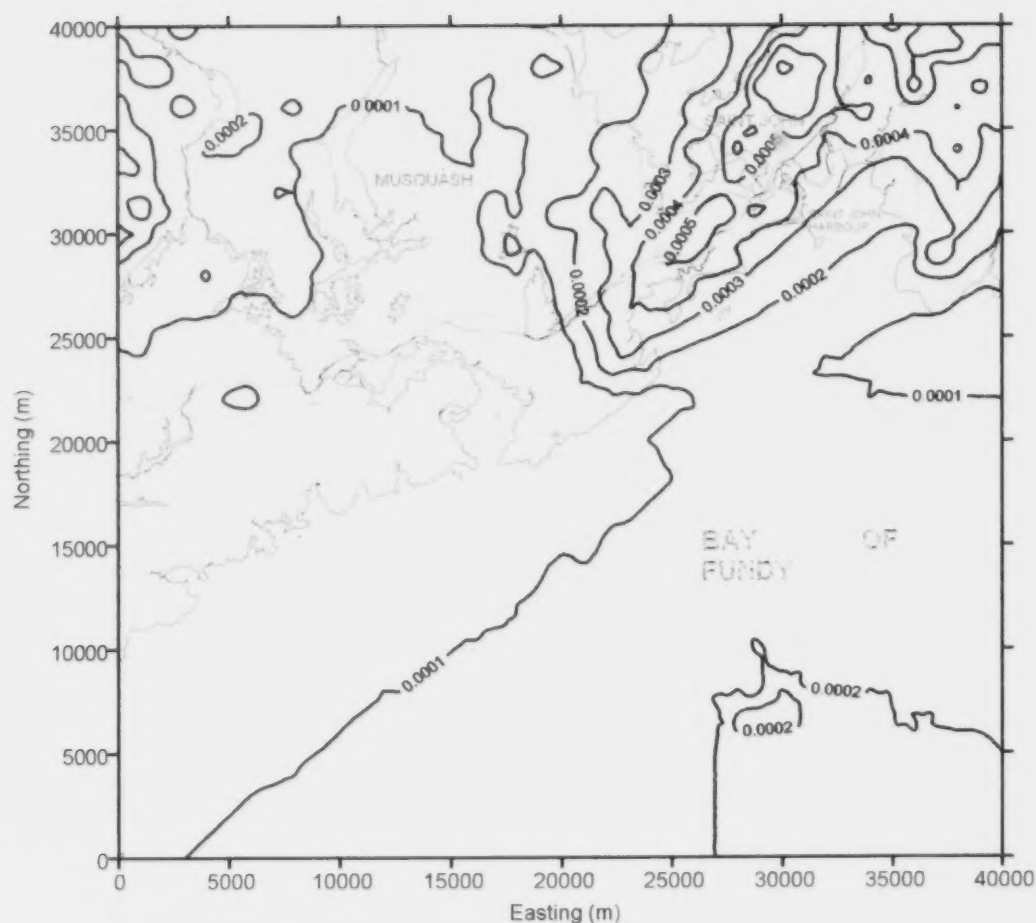
- ★ Coleson Cove
Generating Station
- 1- Concentration, $\mu\text{g}/\text{m}^3$

Job No. 13269-0001



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Figure 2-6
 Predicted Unitized Annual Average Air Concentrations [$\mu\text{g}/\text{m}^3$] / (g/sec)]
 Vapor Form
 Existing Condition - Fuel Oil #6 - High Load Scenario
 Natural Gas - High Load Scenario
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick



LEGEND

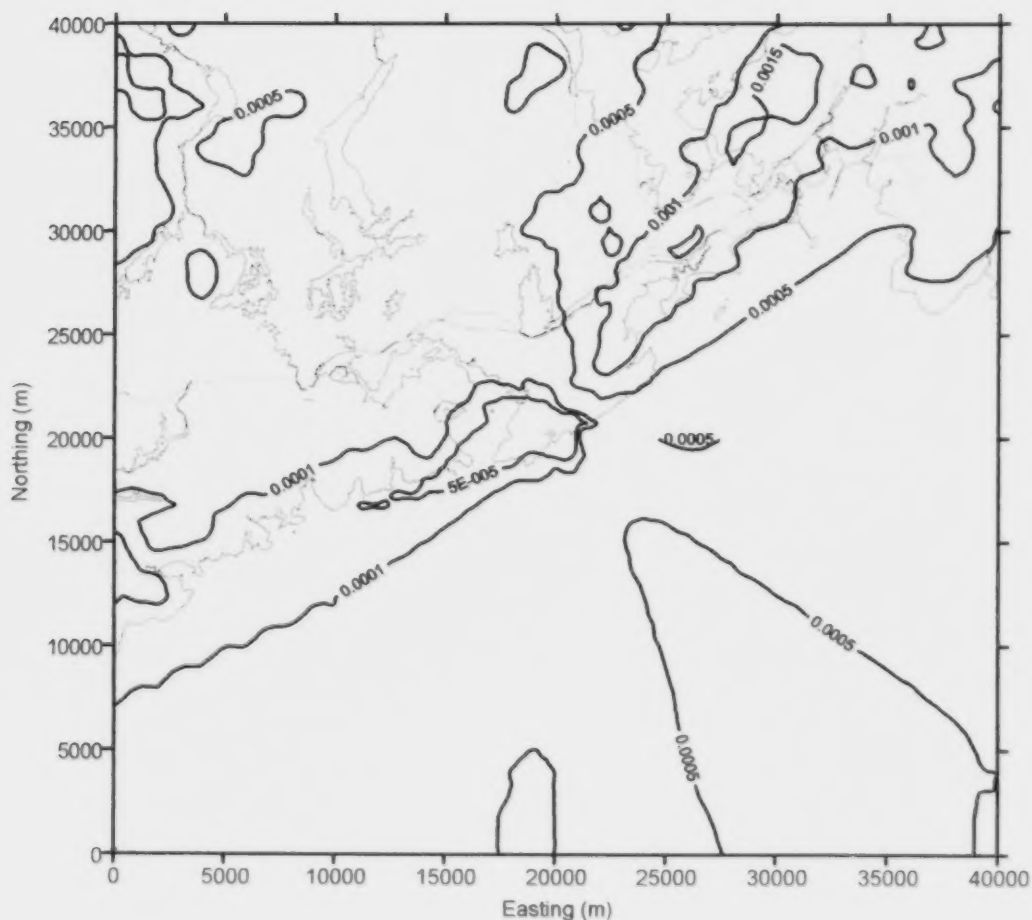
- Coleson Cove
- Generating Station
- 1- Concentration, $\mu\text{g}/\text{m}^3$

Job No. 13269-0001



Jacques Whitford
 Environment Limited

Figure 2-7
Predicted Unitized Annual Average Air Concentrations $[(\mu\text{g}/\text{m}^3) / (\text{g}/\text{sec})]$
Particle-Bound Form
Refurbishment Project - Orimulsion® - High Load Scenario
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick



LEGEND

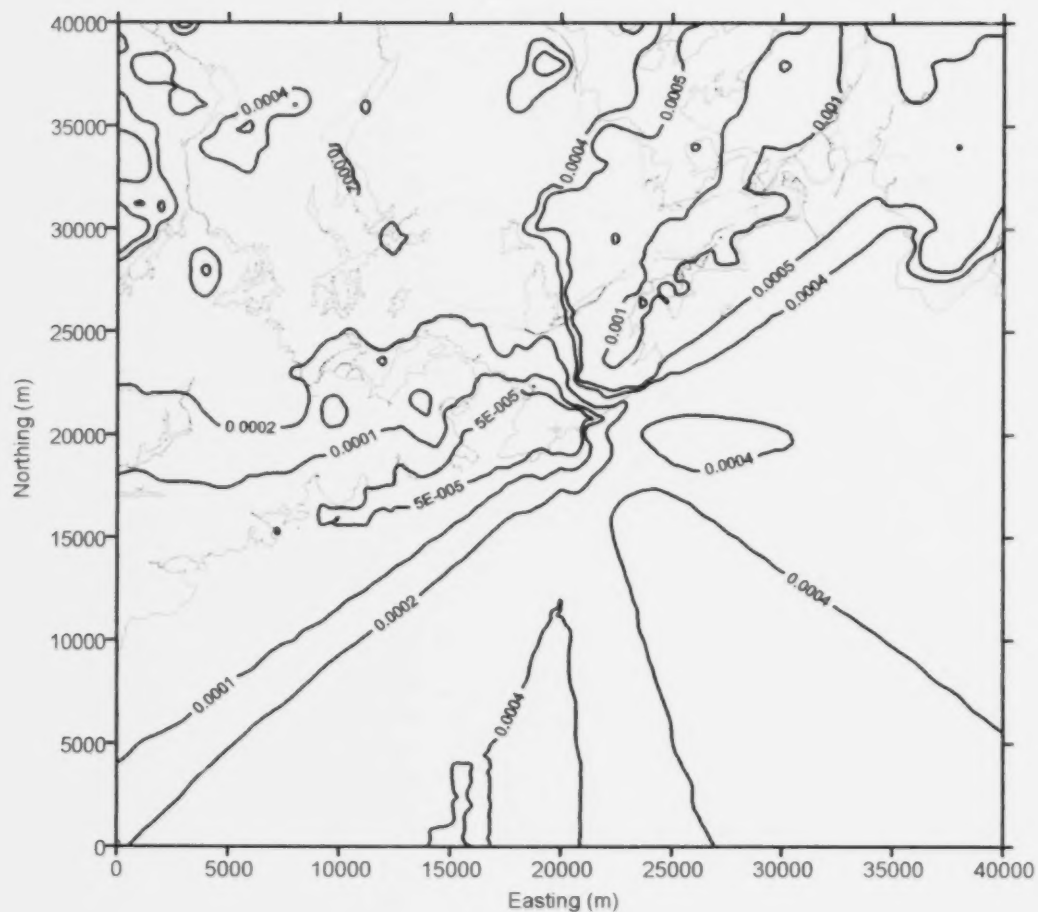
- Coleson Cove
- Generating Station
- 1- Concentration, $\mu\text{g}/\text{m}^3$

Job No. 13269-0001



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Figure 2-8
 Predicted Unitized Annual Average Air Concentrations [$\mu\text{g}/\text{m}^3$ / (g/sec)]
 Particle-Phase Form
 Refurbishment Project - Orimulsion® - High Load Scenario
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick



LEGEND

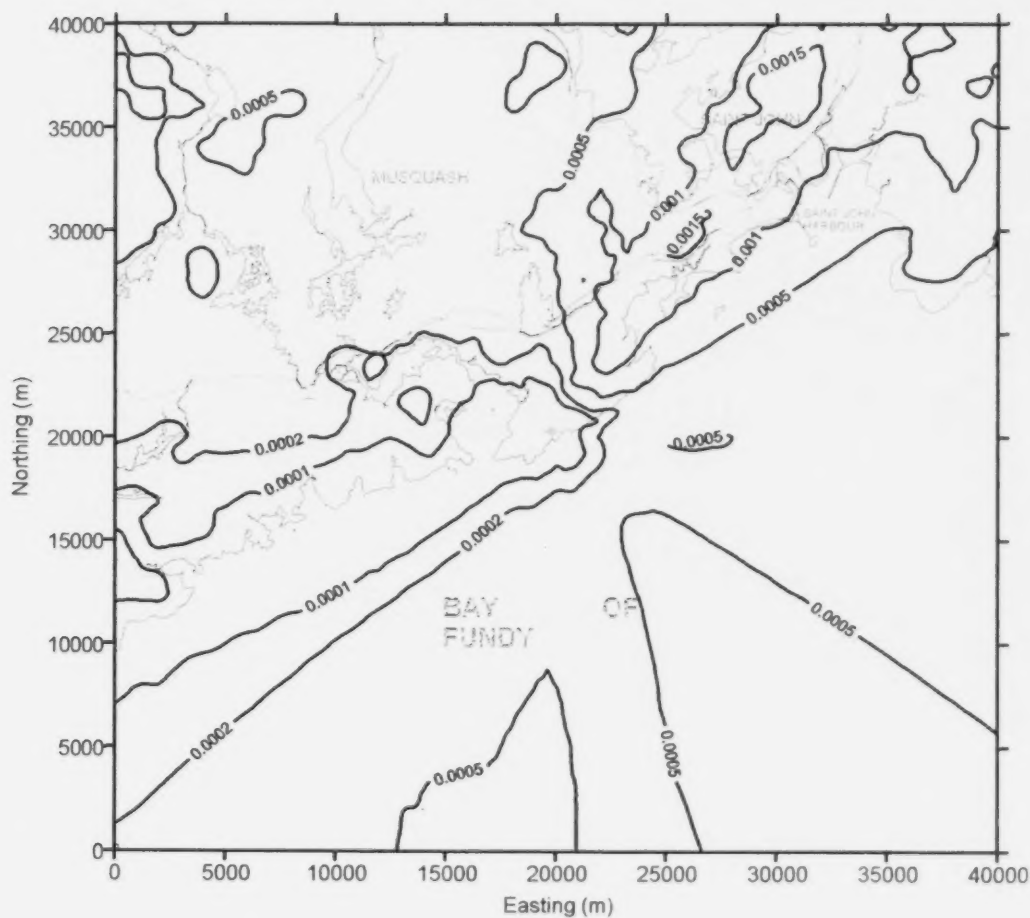
- Coleson Cove
- Generating Station
- 1— Concentration, $\mu\text{g}/\text{m}^3$

Job No. 13269-0001



Jacques Whitford
 Environment Limited

Figure 2-9
Predicted Unitized Annual Average Air Concentrations [$\mu\text{g}/\text{m}^3$ / (g/sec)]
Vapor Form
Refurbishment Project - Orimulsion® - High Load Scenario
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick



LEGEND

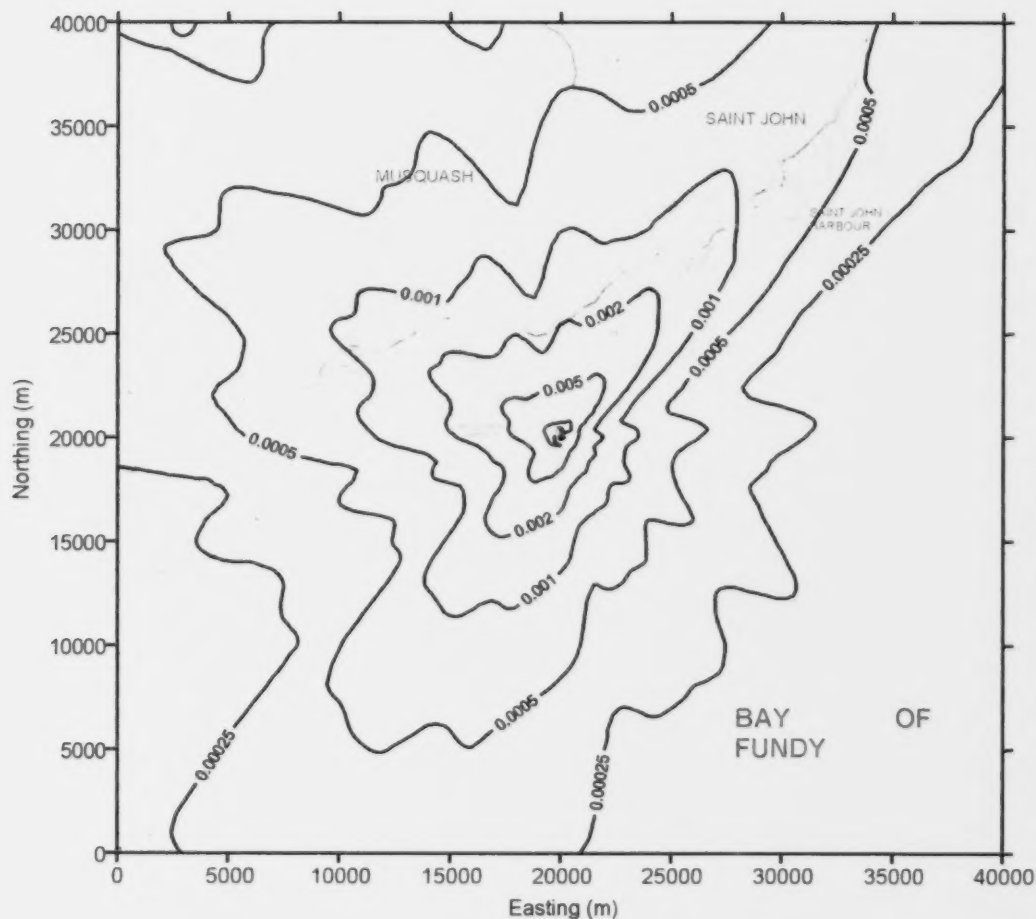
- ★ Coleson Cove
Generating Station
- - - Concentration, $\mu\text{g}/\text{m}^3$

Job No. 13269-0001



Jacques Whitford
Environment Limited

Figure 2-10
 Predicted Unitized Annual Average Total Deposition $[(g/m^2/yr) / (g/sec)]$
 Particle-Bound Form
 Existing Condition - Fuel Oil #6 - High Load Scenario
 Natural Gas - High Load Scenario
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick



LEGEND

Coleson Cove
 Generating Station

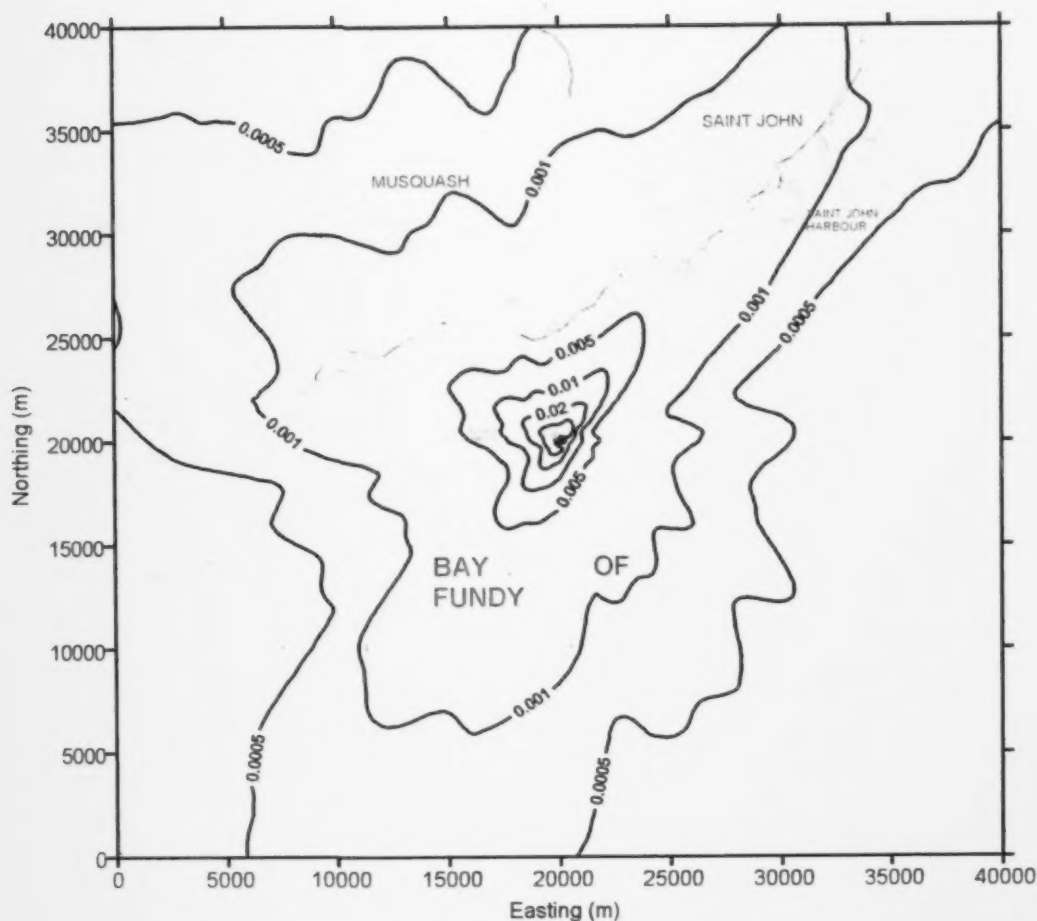
-1- Total Deposition, $g/m^2/yr$

Job No. 13269-0001



Jacques Whitford
 Environment Limited

Figure 2-11
 Predicted Unitized Annual Average Total Deposition [$(\text{g}/\text{m}^2/\text{y}) / (\text{g}/\text{sec})$]
 Particle-Phase Form
 Existing Condition - Fuel Oil #6 - High Load Scenario
 Natural Gas - High Load Scenario
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick



LEGEND

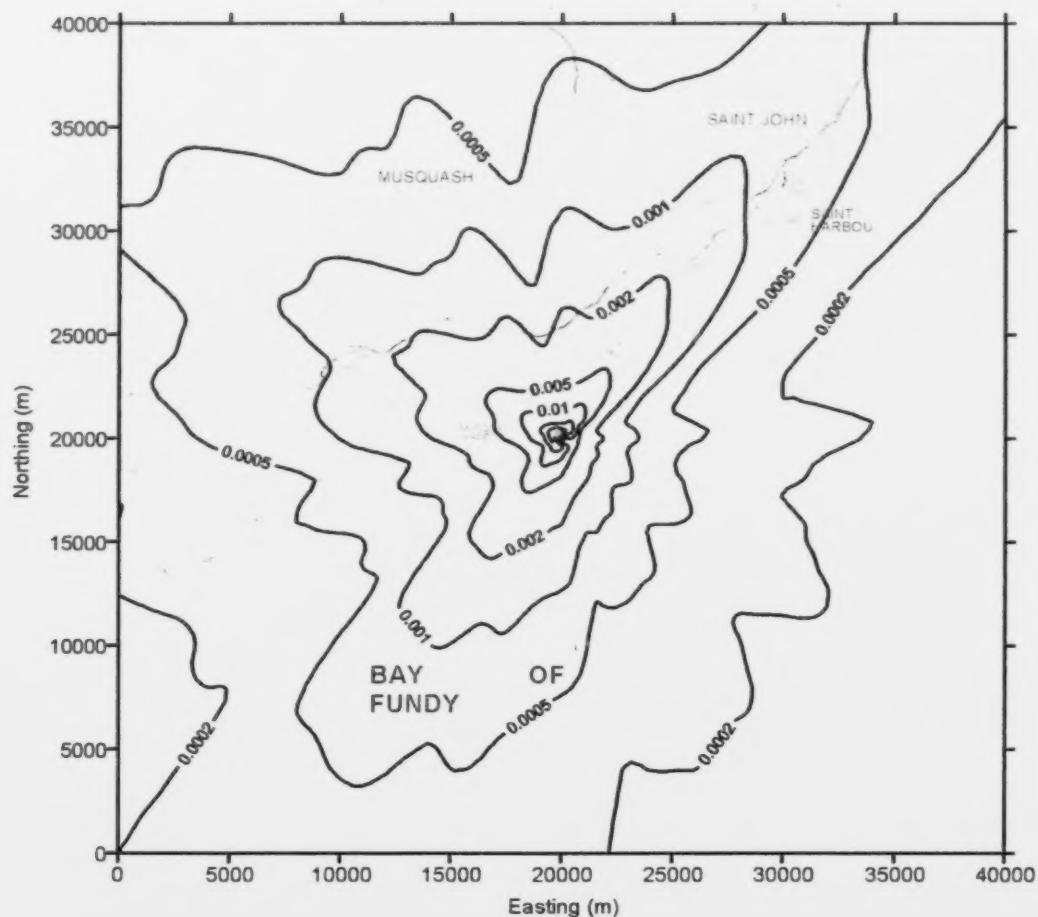
Coleson Cove
 Generating Station
 -1- Total Deposition, $\text{g}/\text{m}^2/\text{yr}$

Job No. 13269-0001



Jacques Whitford
 Environment Limited

Figure 2-12
 Predicted Unitized Annual Average Wet Deposition [(g/m²/y) / (g/sec)]
 Vapor Form
 Existing Condition - Fuel Oil #6 - High Load Scenario
 Natural Gas - High Load Scenario
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick



LEGEND

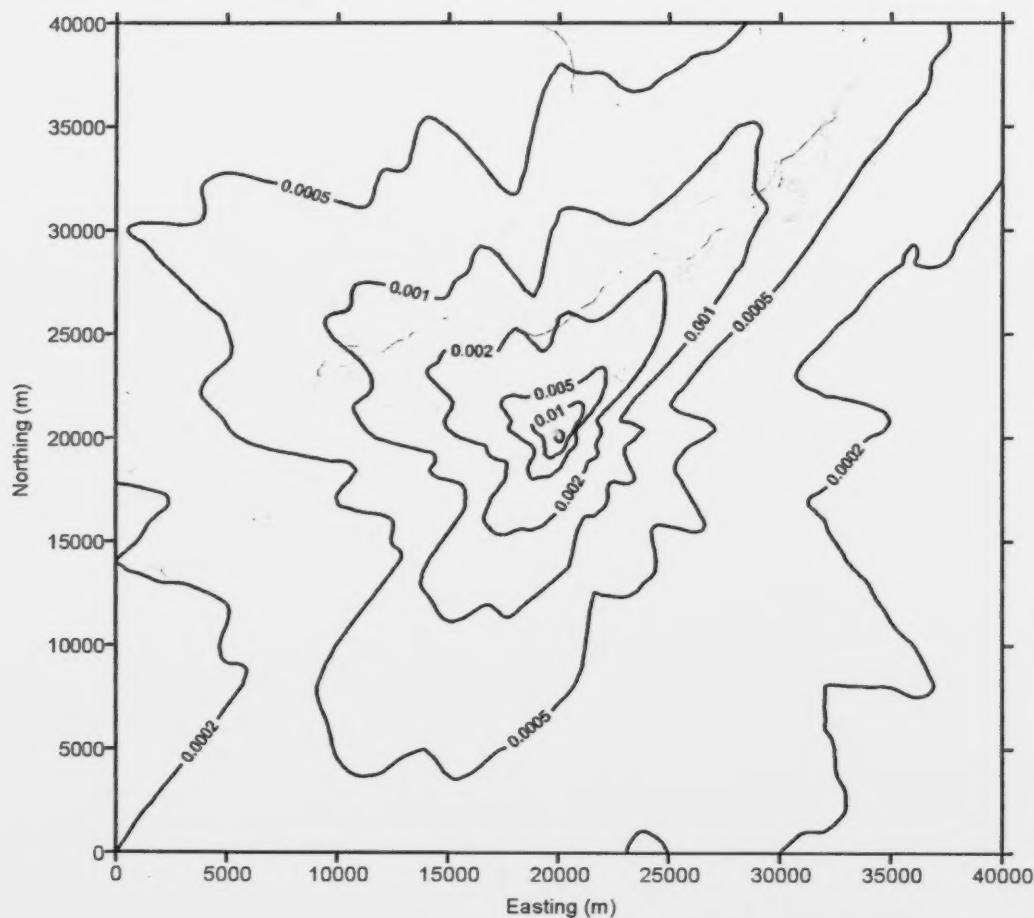
- Coleson Cove Generating Station
- 1- Wet Deposition, g/m²/yr

Job No. 13269-0001



Jacques Whitford
 Environment Limited

Figure 2-13
Predicted Unitized Annual Average Total Deposition $[(\text{g}/\text{m}^2/\text{y}) / (\text{g}/\text{sec})]$
Particle-Bound Form
Refurbishment Project - Orimulsion® - High Load Scenario
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick



LEGEND

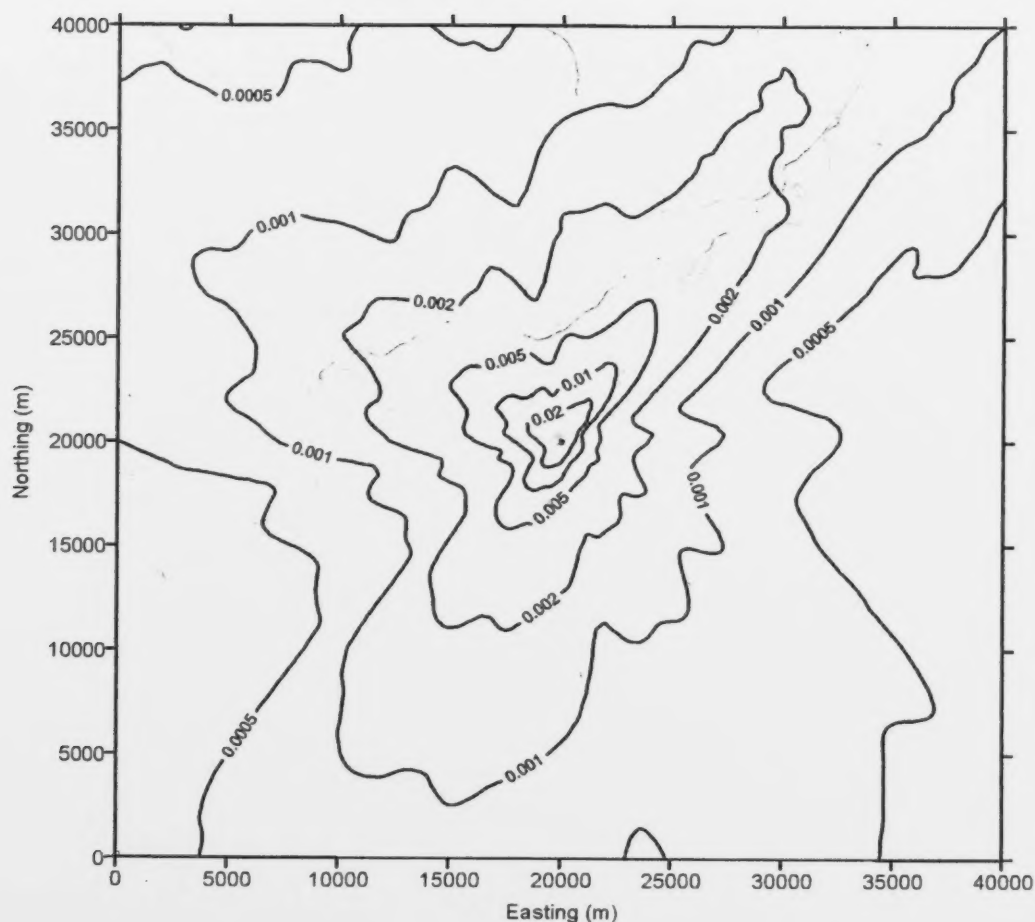
Coleson Cove
 Generating Station
 -1- Total Deposition, $\text{g}/\text{m}^2/\text{yr}$

Job No. 13269-0001



Jacques Whitford
 Environment Limited

Figure 2-14
 Predicted Unitized Annual Average Total Deposition $[(g/m^2/yr) / (g/sec)]$
 Particle-Phase Form
 Refurbishment Project - Orimulsion® - High Load Scenario
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick



LEGEND

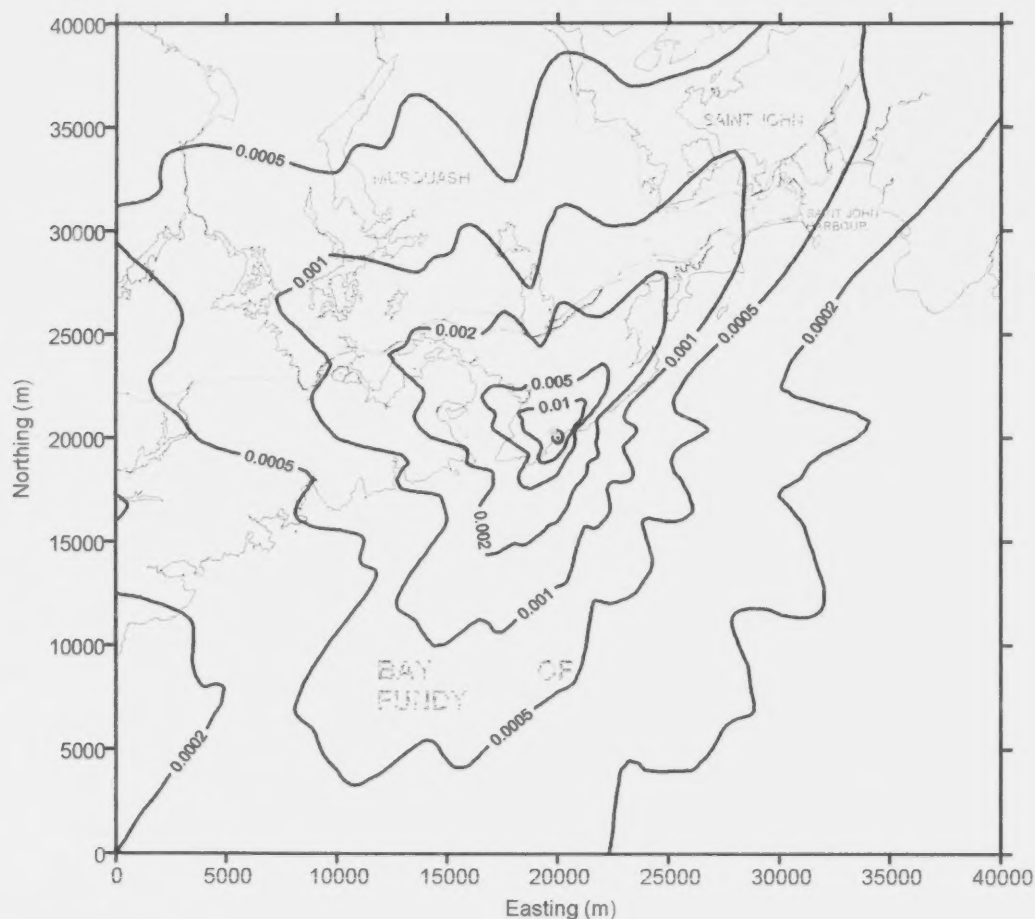
Coleson Cove
 Generating Station
 -1- Total Deposition, $g/m^2/yr$

Job No. 13269-0001



Jacques Whitford
 Environment Limited

Figure 2-15
 Predicted Unitized Annual Average Wet Deposition [(g/m²/y) / (g/sec)]
 Vapor Form
 Refurbishment Project - Orimulsion® - High Load Scenario
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick



LEGEND

- ★ Coleson Cove
Generating Station
- 1- Wet Deposition, g/m²/yr

Job No. 13269-0001



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Environment Limited

3.0 TOXICITY ASSESSMENT

The purpose of the Toxicity Assessment is to identify both the types of adverse health effects a CPC may potentially cause as well as the relationship between the magnitude of CPCs to which receptors may be exposed (dose) and the likelihood of an adverse health effect (response). Adverse health effects are characterized as carcinogenic or noncarcinogenic. Dose-response relationships are defined for oral and inhalation routes of exposure. The results of the Toxicity Assessment, when combined with the results of the Exposure Assessment (Section 4.0), provides a conservative estimate of potential risk.

Section 3.1 describes the approach for developing noncarcinogenic dose-response values and presents the oral and inhalation noncarcinogenic dose-response values used in this risk assessment. The carcinogenic dose-response relationships are discussed in Section 3.2, and the carcinogenic dose-response values used in this risk assessment are presented. Dose-response information used in this risk assessment was obtained from the following sources, in order of preference:

- Health Canada (1996);
- Council of the Canadian Ministries of the Environment (1999), Canadian Environmental Quality Guidelines;
- Atlantic PIRI (1999);
- U.S. EPA's Integrated Risk Information System (U.S. EPA, 2002) or the Health Effects Assessment Summary Tables (U.S. EPA, 1997).

For chemicals that do not have dose-response values listed in these sources, other methods were used to derive dose-response values.

3.1 Noncarcinogenic Dose-Response

Compounds with known or potential noncarcinogenic effects are assumed to have a dose below which no adverse effect occurs, or conversely, above which an effect may be seen. This dose is called the threshold dose. In the laboratory experiments, this dose is known as the No Observed Adverse Effect Level (NOAEL). The lowest dose at which an adverse effect is seen is called the Lowest Observed Adverse Effect Level (LOAEL). Health Canada has used these values to develop Tolerable Daily Intakes (TDIs) for chronic exposures to compounds with potential noncarcinogenic effects. For compounds with potential noncarcinogenic effects, the TDI provides reasonable certainty that if the specified exposure dose is below the threshold, then no noncarcinogenic health effects are expected to occur even if daily exposure were to occur for a lifetime. TDIs are expressed in terms of milligrams of compound per kilogram of body weight per day (mg/kg-day). Table 3-1 summarizes the dose-response information for CPCs with potential noncarcinogenic effects via the inhalation and oral routes of exposure.

Oral TDI values for two CPCs (cadmium and chromium VI) were obtained from the Guidelines for Canadian Drinking Water Quality (CCME, 1999). For two additional compounds (mercury and toluene), oral TDI values were identified from the Canadian Environmental Quality Guidelines (CCME, 1999). Oral TDI values were obtained from Atlantic PIRI (1999) for six compounds (acenaphthylene, benzo(g,h,i)perylene, fluoranthene, naphthalene, phenanthrene, and pyrene). Oral noncarcinogenic dose-response values for ten compounds were obtained from U.S. EPA's IRIS database (U.S. EPA, 2002) (acenaphthene, anthracene, arsenic, beryllium, chromium (total), 1,4-dichlorobenzene, fluorene, formaldehyde, nickel, and vanadium). Oral noncarcinogenic dose-response values for benzene were obtained from a U.S. EPA Region 3 list of toxicity values (U.S. EPA, 2001c). For seven PAH compounds (benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(ah)anthracene, and indeno(1,2,3-cd)pyrene), no oral noncarcinogenic dose-response values were identified. The oral noncarcinogenic dose-response information for naphthalene was used as a surrogate for these seven compounds.

Health Canada (1996) provided an inhalation TDI for nickel. An inhalation TDI value for toluene was obtained from the Canadian Environmental Quality Guidelines (CCME, 1999). Inhalation TDI values were obtained from Atlantic PIRI (1999) for two compounds (acenaphthylene and naphthalene). Inhalation noncarcinogenic dose-response values for three compounds were obtained from U.S. EPA's IRIS database (U.S. EPA, 2002) (mercury, beryllium, and chromium VI). Inhalation noncarcinogenic dose-response values for benzene were obtained from a U.S. EPA Region 3 list of toxicity values (U.S. EPA, 2001c). An inhalation dose-response value was derived from U.S. EPA's National Ambient Air Quality Standard of $1.5 \mu\text{g}/\text{m}^3$ by assuming that a 70 kg adult breathes $20 \text{ m}^3/\text{day}$.

For seven PAH compounds (benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(ah)anthracene, and indeno(1,2,3-cd)pyrene), no inhalation noncarcinogenic dose-response values were identified. The oral noncarcinogenic dose-response information for naphthalene was used as a surrogate for these seven compounds. For an additional 12 compounds (acenaphthene, anthracene, arsenic, benzo(g,h,i)perylene, cadmium, chromium (total), fluoranthene, fluorene, formaldehyde, phenanthrene, pyrene, and vanadium), no inhalation noncarcinogenic dose-response value was identified. For these 12 compounds, the oral noncarcinogenic dose-response value was used as a surrogate.

3.2 Carcinogenic Dose-Response

The underlying assumption of regulatory risk assessment for compounds with known or assumed potential carcinogenic effects is that no threshold dose exists. In other words, it is assumed that a finite level of risk is associated with any dose above zero, theoretically even a single molecule could cause some level of risk. For carcinogenic effects, a two-step evaluation is used, in which the chemical is assigned a weight-of-evidence classification, and then a cancer slope factor (CSF) is calculated. The weight-of-evidence classification is based on the likelihood of the compound being a human carcinogen.

In the second part of the evaluation, CSFs are calculated for compounds that are known or probable human carcinogens. The U.S. EPA's Human Health Assessment Group (HHAG) has developed computerized models that extrapolate observed responses at high doses used in animal studies to predicted responses in humans at the low doses encountered in environmental situations. The models developed by the HHAG assume no threshold and use animal as well as human data to develop an estimate of the carcinogenic potency of a compound. This numerical estimate is referred to as the cancer slope factor. The computerized models assume that carcinogenic dose-response is linear at low doses.

Table 3-2 summarizes the dose-response information for CPCs for this risk assessment assumed to produce potential carcinogenic effects through the oral and inhalation routes of exposure. Carcinogenic dose-response values are expressed in terms of $(\text{mg/kg-day})^{-1}$.

Oral CSF values are available for three compounds (arsenic, benzene, and benzo(a)pyrene) from the Guidelines for Canadian Drinking Water Quality (CCME, 1999). An oral CSF for 1,4-dichlorobenzene was obtained from U.S. EPA's IRIS database (U.S. EPA, 2002). In addition to benzo(a)pyrene, six additional PAH are considered to be potentially carcinogenic. Although cancer slope factors have not been developed for these six PAH, U.S. EPA has developed Toxicity Equivalent Factors (TEFs) that relate the toxicity of these PAH to that of benzo(a)pyrene, for which a CSF is available from CCME (1999). The TEFs range from 0.001 to 1, and are intended to be multiplied by the cancer slope factor for benzo(a)pyrene to provide an estimate of the carcinogenic potency of the other six PAH. Table 3-3 lists the TEFs used to evaluate potentially carcinogenic PAH in this risk assessment. Table 3-2 shows the estimated CSFs for all potentially carcinogenic PAH, based on the benzo(a)pyrene CSF and the TEFs.

Health Canada (1996) provides inhalation CSFs for four compounds (arsenic, cadmium, chromium VI, and nickel). Inhalation CSF values were obtained from Atlantic PIRI (1999) for two compounds (benzene and benzo(a)pyrene). Inhalation CSF values for three compounds (beryllium, 1,4-dichlorobenzene, and formaldehyde) were obtained from U.S. EPA's IRIS database (U.S. EPA, 2002). Inhalation CSFs for the six potentially carcinogenic PAH compounds for which no CSF has been developed were estimated in the same manner as the oral CSF, using the inhalation CSF for benzo(a)pyrene and the TEFs shown in Table 3-3.

Table 3-1
Dose-Response Information for Compounds with Potential Noncarcinogenic Effects
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Chemical	Oral TDI (mg/kg-day)	Source	Target Organ	Inhalation TDI (mg/kg-day)	Source	Target Organ
Mercury, Elemental	3.00E-04	(d)	Central nervous system	8.57E-05	(b)	Nervous system
Mercury, Methyl	3.00E-04	(d)	Central nervous system	1.00E-04	(b)	Nervous system
Mercury, Divalent	3.00E-04	(d)	Central nervous system, immune system	3.14E-04	(b)	Nervous system
1,4-Dichlorobenzene	2.30E-01	(b)	Liver	2.29E-01	(b)	Liver
Acenaphthene	6.00E-02	(b)	Liver	6.00E-02	(e)	Liver
Acenaphthylene	4.00E-03	(c)	Liver	6.00E-02	(c)	
Anthracene	3.00E-01	(b)	Liver	3.00E-01	(e)	Liver
Arsenic	3.00E-04	(b)	Skin	3.00E-04	(e)	Skin
Benzene	3.00E-03	(f)	---	1.70E-03	(f)	---
Benzo(a)anthracene	2.00E-02	(g)	Body weight	1.60E-02	(g)	---
Benzo(a)pyrene	2.00E-02	(g)	Body weight	1.60E-02	(g)	---
Benzo(b)fluoranthene	2.00E-02	(g)	Body weight	1.60E-02	(g)	---
Benzo(k)fluoranthene	2.00E-02	(g)	Body weight	1.60E-02	(g)	---
Benzo(g,h,i)perylene	4.00E-03	(c)	---	4.00E-03	(e)	---
Beryllium	2.00E-03	(b)	Gastro-intestinal tract	5.70E-06	(b)	---
Cadmium	8.20E-04	(a)	Kidney	8.20E-04	(e)	Kidney
Chromium	1.50E+00	(b)	Kidney	1.50E+00	(e)	Kidney
Chromium VI	1.10E-03	(a)	Kidney	2.90E-05	(b)	---
Chrysene	2.00E-02	(g)	Body weight	1.60E-02	(g)	---
Dibenzo(a,h)anthracene	2.00E-02	(g)	Body weight	1.60E-02	(g)	---
Fluoranthene	4.00E-02	(c)	Kidney, Liver, Blood	4.00E-02	(e)	Kidney, Liver, Blood
Fluorene	4.00E-02	(b)	Erythrocytes	4.00E-02	(e)	Gastro-intestinal tract
Formaldehyde	2.00E-01	(b)	Gastro-intestinal tract, Body weight	2.00E-01	(e)	Body weight
Indeno(1,2,3-cd)pyrene	2.00E-02	(g)	Body weight	1.60E-02	(g)	---
Lead	1.85E-03		Central nervous system	4.30E-04	(i)	---
Napthalene	2.00E-02	(c)	Body weight	1.60E-02	(c)	
Nickel	2.00E-02	(b)	Organ weight	5.70E-06	(h)	---
Phenanthrene	4.00E-03	(c)	Liver	4.00E-03	(e)	Liver
Pyrene	3.00E-02	(c)	Kidney	3.00E-02	(e)	Kidney
Toluene	2.23E-01	(d)	Liver, Kidney	1.25E+00	(d)	
Vanadium	9.00E-03	(b)	Hair	9.00E-03	(e)	Hair

Notes:

- (a) Guidelines for Canadian Drinking Water Quality. http://www.hc-sc.gc.ca/ehp/ehd/catalogue/bch_pubs/dwgsup_doc/dwgsup_doc.htm
- (b) US EPA 2002. Integrated Risk Information System (IRIS).
- (c) Atlantic PIRI. April 1999. Atlantic RBCA Reference Documentation for Petroleum Impacted Sites. Version 1.0.
- (d) TDI (Tolerable Daily Intake) from: Canadian Council of Ministers of the Environment (CCME). 1999. Canadian Environmental Quality Guidelines.
- (e) Oral TDI or Reference Dose used as a surrogate
- (f) US EPA 2001.
- (g) Naphthalene values used as surrogates
- (h) Health Canada. 1996.
- (i) Converted from a U.S. EPA National Ambient Air Quality Standard of 1.5 ug/m³ (43 FR 46258, Oct. 5, 1978), assuming a 70 kg person breathes 20 m³/day.

Table 3-2
Dose-Response Information for Compounds with Potential Carcinogenic Effects
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Chemical	Oral CSF (mg/kg-day) ⁻¹	Source	Target Organ	Inhalation CSF (mg/kg-day) ⁻¹	Source	Target Organ
Mercury, Elemental	ND	---	NA	ND	---	NA
Mercury, Methyl	ND	---	NA	ND	---	NA
Mercury, Divalent	ND	---	NA	ND	---	NA
1,4-Dichlorobenzene	2.40E-02	(b)	Liver	2.40E-02	(b)	Liver
Acenaphthene	ND	---	NA	ND	---	NA
Acenaphthylene	ND	---	NA	ND	---	NA
Anthracene	ND	---	NA	ND	---	NA
Arsenic	1.67E+00	(a)	Skin, lung	2.24E+01	(e)	Respiratory system
Benzene	2.90E-02	(a)	Blood	2.90E-02	(c)	Blood
Benzo(a)anthracene	2.30E-01	(a,f)	Blood	3.10E-01	(c)	
Benzo(a)pyrene	2.30E+00	(a)	Stomach, larynx, esophagus	3.10E+00	(c)	
Benzo(b)fluoranthene	2.30E-01	(a,f)	Blood	3.10E-01	(c)	
Benzo(k)fluoranthene	2.30E-02	(a,f)	Blood	3.10E-02	(c)	
Benzo(g,h,i)perylene	ND	---	NA	ND	---	NA
Beryllium	ND	---	NA	8.40E+00	(b)	Lung
Cadmium	ND	---	NA	3.43E+01	(e)	Lung
Chromium	ND	---	NA	ND	---	NA
Chromium VI	ND	---	NA	2.65E+02	(e)	Lung
Chrysene	2.30E-03	(a,f)	Blood	3.10E-03	(c)	
Dibenzo(a,h)anthracene	2.30E+00	(a,f)	Blood	3.10E+00	(c)	
Fluoranthene	ND	---	NA	ND	---	NA
Fluorene	ND	---	NA	ND	---	NA
Formaldehyde	ND	---	NA	4.50E-02	(b)	Nasal cavity
Indeno(1,2,3-cd)pyrene	2.30E-01	(a,f)	Blood	3.10E-01	(c)	
Lead	ND	---	NA	ND	---	NA
Napthalene	ND	---	NA	ND	---	NA
Nickel	ND	---	NA	4.38E+00	(e)	Lung
Phenanthrene	ND	---	NA	ND	---	NA
Pyrene	ND	---	NA	ND	---	NA
Toluene	ND	---	NA	ND	---	NA
Vanadium	ND	---	NA	ND	---	NA

Notes:

(a) Guidelines for Canadian Drinking Water Quality. http://www.hc-sc.gc.ca/ehp/ehd/catalogue/bch_pubs/dwgsup_doc/dwgsup_doc.htm

(b) US EPA, 2002. Integrated Risk Information System (IRIS).

(c) Atlantic PIRI, 1999. Atlantic RBCA Reference Documentation for Petroleum Impacted Sites. Version 1.0.

(d) Canadian Council of Ministers of the Environment (CCME). 1999. Canadian Environmental Quality Guidelines.

(e) Health Canada. 1996.

(f) U.S. EPA, 1994.

ND: Not determined.

NA: Not Applicable

Table 3-3
 Toxic Equivalent Factors for Potentially Carcinogenic PAH
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Chemical	TEF (a)
Benzo(a)pyrene	1.0
Benzo(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Chrysene	0.001
Dibenzo(a,h)anthracene	1.0
Indeno(1,2,3-cd)pyrene	0.1

Notes:

(a) U.S. EPA (1994).

4.0 EXPOSURE ASSESSMENT

This section identifies the type and magnitude of potential exposures to CPCs which receptors may experience as a result of exposure to emissions from the CCGS. First, potential receptors are identified, based on conditions present at the facility and surrounding area. Next, potential routes of exposure are identified for each receptor, based on information about activities which typically occur in the area.

Section 4.1 of the Exposure Assessment describes the study area. Based on this information, receptors are identified in Section 4.2. The potential exposure pathways by which these receptors may be exposed are identified in Section 4.3. Section 4.4 describes the methodology used to estimate exposure point concentrations, or the magnitude of compounds which a receptor may contact in a given environmental medium. Finally, a discussion of the methodology used to estimate exposure doses is explained in Section 4.5. This section also includes a discussion of the exposure parameters assumed for each receptor and exposure pathway.

4.1 Description of the Study Area

The CCGS is located in Lorneville, New Brunswick, in Coleson Cove, immediately inland from the Bay of Fundy. Figure 2-1 depicts the facility and surrounding location. The facility property extends to the northeast to Burchill Road. The facility is surrounded on the north and west by parkland and to the east and south by the Bay of Fundy. The land immediately to the northeast of the facility is parkland owned by the Province of New Brunswick Department of Economic Development and Tourism.

Three interconnected lakes (Spruce Lake, Menzies Lake, and Ludgate Lake) are located about 5 km north of the facility. These lakes serve as the source of drinking water for the city of Saint John. The closest location to the facility where farming could occur is located about 20 km north-northeast of the facility. The City of Saint John, the closest major residential area, is about 15 km northeast of the facility. The closest residence to the facility is located approximately 2 miles northeast of the facility.

The risk assessment study area is defined as a zone with a radius of approximately 20 km around the facility. This study area is shown in Figure 2-3.

4.2 Identification of Receptors

The risk assessment guidance requires that risks be evaluated for three groups of people (residents, farmers, and fishers) who may or may not actually live in the study area around the facility that is the subject of the risk assessment. This is required so that all of the ways that facility emissions could affect human health are evaluated. The guidance requires that these groups of people are assumed to be located in areas defined as locations of maximum impact where it is reasonable to anticipate that they could be exposed to facility emissions. AMEC is

aware that little freshwater fishing occurs in Spruce Lake, Menzies Lake, and Ludgate Lake and that there is a fish advisory in effect that recommends against the consumption on many species of freshwater fish in the water bodies of New Brunswick. AMEC is also aware that little, if any, farming occurs within the study area. However, standard risk assessment guidance requires that exposures due to fishing and farming be evaluated because such activities *could* happen in the future even in areas where they do not now occur.

After evaluating the results of the air dispersion and deposition modeling, AMEC identified a location directly adjacent to the property boundary as the location for the maximally affected hypothetical resident (and recreational fisher) and a location northwest of the City of Saint John as the location for the maximally affected hypothetical farmer. In addition, the City of Saint John was identified as another location for which risks would be evaluated for residents and recreational fishers. Each of these groups of people are called "receptors" in this risk assessment. The five receptors identified are summarized as follows:

- Resident living at the closest hypothetical residential location
- Resident living in Saint John
- Farmer living at the farm northeast of the facility
- Recreational fisher living at the closest hypothetical residential location
- Recreational fisher living in Saint John

As mentioned above, the land surrounding the facility in the downwind direction (northeast) is parkland owned by the Province of New Brunswick Department of Economic Development and Tourism. Although this location cannot currently be developed for residential use, it is conceivable that in the future, such development could occur. Although no residences exist at the eastern property boundary of the facility, the risk assessment includes an evaluation of this area as the maximum hypothetical residence. The maximum hypothetical resident was assumed to reside at this location. To estimate the unitized air concentrations and deposition rates necessary for modeling exposure point concentrations, the results of air quality modeling at three receptor nodes along the property boundary [(X=100m,Y=600m), (X=200,Y=400m), and (X=300m,Y=300m)] were averaged to represent conditions at this location. This location is the location of the maximum wet deposition from the facility. Wet deposition drives the results of the risk assessment. Thus, this location is the location of highest total risk. However, unitized air concentrations and unitized dry deposition rates at this location were estimated to be zero in air quality modeling. As a conservative measure, a value of 0.00001 (ug/m³)/(g/sec) was used to represent unitized air concentrations at this location, and a value of 0.00001 (g/m²/yr)/(g/sec) was used to represent unitized dry deposition at this location. These are the lowest non-zero results produced by the air quality model.

The Saint John resident was assumed to live in the City of Saint John, approximately 15 km northeast of the facility. To estimate the unitized air concentrations and deposition rates necessary for modeling exposure point concentrations, the results of air quality modeling at the receptor nodes "covering" the City of Saint John were averaged to represent conditions at this location. Figure 2-3 identifies the receptor nodes used to represent the City of Saint John.

The farmer was assumed to reside in the area of the closest actual farming location in the direction of maximum facility impacts. A review of land use in with the study area indicates that the peninsula north of Saint John is the closest farming to the facility. This location is 20 km north-northeast of the facility. To estimate the unitized air concentrations and deposition rates necessary for modeling exposure point concentrations, the results of air quality modeling at the receptor nodes "covering" the farming location were averaged to represent conditions at this location. Figure 2-3 identifies the receptor nodes used to represent the farming location.

The recreational fisher was assumed to reside either at the closest residential location or in the City of Saint John, and to catch fish from the Spruce/Menzies/Ludgate Lakes complex. To estimate the unitized air concentrations and deposition rates necessary for modeling concentrations in the watershed and waterbody, the results of air quality modeling at the receptor nodes "covering" the watershed and the water body were averaged to represent conditions at this location. Figure 2-3 identifies the receptor nodes used to represent these areas.

Both child and adult receptors were evaluated for all scenarios.

4.3 Description of Potential Exposure Pathways

Potential exposure pathways are the mechanisms by which the receptors in the study area may be exposed to compounds emitted from the proposed facility. According to U.S. EPA (1989), four elements must be present in order for a potential human exposure pathway to be complete:

- a source and mechanism of compound release to the environment (in this case, emissions from the CCGS stack);
- an environmental transport medium (in this case, air);
- an exposure point, or point of potential contact with the potentially impacted medium; and
- a receptor (i.e., a person) with a route of exposure at the point of contact.

Both direct and indirect exposure pathways were evaluated. Direct exposure pathways involve a receptor's direct contact with a compound in an environmental medium such as air, water or soil. The direct pathways examined in this risk assessment include:

- inhalation of particulate and gaseous compounds;
- incidental ingestion of soil; and
- ingestion of drinking water.

Indirect exposure pathways involve exposure to compounds through the food chain. For example, people may be indirectly exposed to compounds by eating fish that have been exposed to compounds in water or sediments. The indirect pathways evaluated in this risk assessment include:

- consumption of produce;
- consumption of fish;
- consumption of beef;

- consumption of pork;
- consumption of poultry;
- consumption of eggs; and
- consumption of dairy products.

The risk assessment assumed that exposure could occur through the following direct and indirect pathways:

- inhalation of emissions from the facility;
- contact with soil while outdoors;
- consumption of produce grown in backyard gardens or farms;
- consumption of fish caught from Spruce/Ludgate/Menzies Lakes;
- consumption of beef from the farm located northeast of the facility;
- consumption of dairy products from the farm located northeast of the facility;
- consumption of pork from the farm located northeast of the facility;
- consumption of poultry from the farm located northeast of the facility;
- consumption of eggs from the farm located northeast of the facility; and
- drinking water exposure to water from the Spruce/Ludgate/Menzies Lakes.

However, not all pathways are evaluated for all receptors. Potential pathways evaluated for the resident receptors included:

- inhalation of emissions from the facility;
- contact with soil while outdoors;
- consumption of produce grown in backyard gardens; and
- drinking water exposure to water from the Spruce/Ludgate/Menzies Lakes.

Potential pathways evaluated for the fisher receptors included:

- inhalation of emissions from the facility;
- contact with soil while outdoors;
- consumption of produce grown in backyard gardens;
- consumption of fish caught from Spruce/Ludgate/Menzies Lakes; and
- drinking water exposure to water from the Spruce/Ludgate/Menzies Lakes.

Potential pathways for the farmer receptor included:

- inhalation of emissions from the facility;
- contact with soil while outdoors;
- consumption of produce grown at the farm;
- consumption of beef from the farm;
- consumption of dairy products from the farm;
- consumption of pork from the farm;
- consumption of poultry from the farm; and

- consumption of eggs from the farm.

The amount of exposure through different pathways varies depending on the receptor's location and activities (discussed in Sections 4-4 and 4-5). Certain pathways do not depend on the location where a receptor is assumed to reside. Such pathways include consumption of fish and drinking water ingestion. The extent of exposure via other pathways (inhalation, soil ingestion, consumption of produce) depends on the location where the receptor is assumed to reside. Only the farmer was assumed to be exposed via consumption of beef, dairy products, pork, poultry, and eggs. Only the fisher was assumed to be exposed via consumption of fish.

The input parameters used to quantify each exposure pathway are described in Section 4.5.

4.4 Estimation of Exposure Point Concentrations

This section describes the mechanisms by which compounds emitted from the CCGS are transported through the environment (air, soil, and water) to points of exposure where receptors may contact them. The modeling techniques used to estimate exposure point concentrations in environmental media affected by facility emissions are also summarized in this section.

Compound concentrations in each medium at each location are also presented in this section.

4.4.1 Ambient Air

The receptors evaluated in this risk assessment may be exposed via inhalation to compounds emitted from the proposed facility. Receptors are assumed to be exposed to all compounds expected to be emitted from the facility via the inhalation route of exposure. In addition, receptors are also assumed to be exposed to all compounds, including all metals and semivolatile organic compounds, as a result of deposition onto soil, water, or vegetation in the study area. All compounds may be available for human consumption via the food chain.

Concentrations of gaseous, particle-phase, and particle-bound compounds in air have been determined using air dispersion modeling. Section 2.3 describes the methodology used to estimate the concentrations of compounds in ambient air. Concentrations of compounds in ambient air were predicted for the following locations:

- Maximum hypothetical residence;
- City of Saint John; and
- Farm closest to the facility.

Deposition modeling has been performed using the modeling approach, as described in Section 2.3. As previously stated, all compounds are assumed to deposit on available surfaces. Predicted concentrations in air at the identified locations resulting from emissions from the CCGS can be derived from Tables 2-7 to 2-9 (for the three sets of operating conditions) using the unit emission rates reported in Table 2-2 (for the three sets of operating conditions), as follows:

$$A = B \times [C \times D + (1 - C) \times E]$$

where:

A = Compound Concentration in Air ($\mu\text{g}/\text{m}^3$)

B = Compound Emission Rate (g/sec)

C = Fraction of Compound Air Concentration in Vapor Form (unitless)

D = Unitized Air Concentration from Vapor Phase ($\mu\text{g}/\text{m}^3$)/(g/sec)

E = Unitized Air Concentration from Particle Phase or Particle-Bound ($\mu\text{g}/\text{m}^3$)/(g/sec)

Table 4-1 shows chemical-specific parameter values used to calculate EPCs, including the fraction of the air concentration in vapor phase. For mercury modeling, U.S. EPA recommends assuming that 0.2% of the total mercury emissions are elemental mercury and 48% of the total mercury emissions are divalent mercury. Methyl mercury is not assumed to exist in air.

Table 4-2 shows the predicted concentrations of compounds in air at the maximum hypothetical residential location, the City of Saint John, and the Farm in the Existing Operation - Fuel Oil #6 - High Load Scenario. Predicted concentrations in air at the maximum residential location, the City of Saint John, and the Farm in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 4-3. Table 4-4 shows the predicted concentrations of compounds in air at the maximum residential location, the City of Saint John, and the Farm in the Natural Gas - High Load Scenario.

4.4.2 Soil

Many of the compounds emitted from the proposed facility are assumed to be bound to particulates or are assumed to be emitted in particulate phase, and deposition of these particulates onto the soil surface is assumed to take place during the entire lifespan of the facility. Vapor phase chemicals are subject to wet deposition phenomena.

Soil concentrations due to CPC emissions were calculated at four locations in the vicinity of the facility. These included a specific farm estimated to receive the highest rate of compound deposition, the area comprising the City of Saint John, the closest hypothetical residential location in the downwind direction, and the area comprising the watershed for the Spruce/Ludgate/Menzies Lakes complex.

The calculation of soil concentration at a location depends on the deposition rate of each chemical at that location, as well as the properties of each chemical that control its loss from the soil. Loss of concentration from soil can be from chemical degradation, volatilization, as well as from leaching due to infiltration.

Chemical concentrations in soil were estimated from predicted deposition rates using U.S. EPA methodology (U.S. EPA, 1998a), as corrected and amended in subsequent U.S. EPA publications, which include:

- U.S. EPA Region 6 Mercury Fate and Transport Addendum (U.S. EPA, 2000)
- U.S. EPA Corrections to the HHRAP (U.S. EPA, 1999b)

The guidance materials were used to estimate CPC concentrations in soil. As recommended by U.S. EPA, different algorithms were used to estimate CPC concentrations for the purposes of estimating potential carcinogenic and noncarcinogenic risk. The algorithms for estimating CPC concentrations for estimating potential carcinogenic effects calculated the concentration averaged over the exposure duration (30 years), and the algorithm for estimating potential noncancer effects calculated the maximum annual soil concentration.

Estimated CPC concentrations in soil assume annual average deposition rates and loss of CPC from the soil via leaching, runoff, erosion, degradation, and volatilization. U.S. EPA (1998a) provides estimates of biotic and abiotic degradation in soil. CPC loss via surface runoff, erosion, and leaching are estimated using equations supplied in U.S. EPA (1998a).

The calculations assume that the facility is operating for 30 years. Concentrations were calculated for two chemical mixing depths (1 cm and 20 cm). The 1 cm depth is referred to as surface soil and used to estimate exposure via incidental soil ingestion, uptake into forage vegetation, and for estimating erosion and runoff. The 20 cm depth is referred to as subsurface soil and used to estimate compound concentrations in garden produce, grain, and silage vegetation.

Table 4-5 shows the predicted concentrations of compounds in 1 cm and 20 cm soil used to evaluate both noncancer and cancer risks at the maximum hypothetical residential location, the City of Saint John, and the Farm in the Existing Operation - Fuel Oil #6 - High Load Scenario. Predicted concentrations in 1 cm and 20 cm soil used to evaluate both noncancer and cancer risks at the maximum hypothetical residential location, the City of Saint John, and the Farm in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 4-6. Table 4-7 shows the predicted concentrations of compounds in 1 cm and 20 cm soil used to evaluate both noncancer and cancer risks at the maximum hypothetical residential location, the City of Saint John, and the Farm in the Natural Gas - High Load Scenario.

4.4.3 Surface Water

Just as emitted compounds may deposit on soil surfaces, compounds may also deposit onto the surface of a water body, such as the Spruce/Ludgate/Menzies Lakes complex. Soil in the designated watershed area of the surface water bodies onto which compounds have deposited may be eroded from nearby hillsides. Individuals who live within the study area may catch and eat fish from the Lakes complex, which have taken up compounds that have deposited on the surface of the lakes or runoff into the lakes. In addition, it was assumed that residents may obtain their drinking water from the Lakes complex. U.S. EPA guidance (1998a) and accompanying Errata (U.S. EPA, 1999b) and Mercury Example Calculation (U.S. EPA, 2000) were used to estimate concentrations of compounds in surface water in the Lakes complex as a result of direct deposit onto the water surface and loading via soil erosion. Data on the physical characteristics of

the Lakes complex (surface area of the water body, depth of the water body, surface area of the watershed) were obtained from Jacques Whitford Environment Limited (JWEL, 2002a).

Estimates of the surface area of the Lakes were made using topographic maps. The combined surface area of Spruce Lake, Ludgate Lake, and Menzies Lake was estimated to be approximately 5,600,279 m². Since the Lakes watershed is almost entirely undeveloped, it was assumed to have an impervious surface area of 1% of the total watershed area. The pervious watershed area was estimated to be 20,400,000 m² (JWEL, 2002a). The average depth of the Lakes complex was estimated to be 20 m (JWEL, 2002a). Because the Lakes complex includes outflow to a reservoir system that holds the drinking water supply for the Saint John area, an average annual flow of 0.58 m³/sec was used (JWEL, 2002a).

The average annual precipitation in the Saint John area is 144 cm/year (JWEL, 2002a). The average annual surface runoff at the facility was estimated to be 102.2 cm/year, and the average annual evapotranspiration value of 52.8 cm/year were estimated (JWEL, 2002a). An average annual irrigation value of zero was assumed.

The Universal Soil Loss Equation (USLE) and a sediment delivery ratio were employed to estimate rates of erosion. The calculation of surface water concentrations assumed a sediment mass balance (that is, sediments do not accumulate and are not lost from the water body) and equilibrium conditions between surficial sediments and the water column. Concentrations of CPCs in the water column and in benthic sediments were calculated, based upon the estimated concentrations in the total water body and the fraction of water body concentration in the water column and benthic sediments, respectively. CPC loads to the water body were based on average deposition rates to land and water surfaces in the Lakes complex watershed.

The USLE requires several area-specific input values to predict soil loss. The rainfall-erosivity factor is a measure of the energy from a storm event and the associated sediment erosion generated. The rainfall-erosivity factor used for the Saint John area is 100 (JWEL, 2002a).

Compound concentrations in surface water were estimated from predicted deposition rates using U.S. EPA methodology (U.S. EPA, 1998a, 1999b, 2000). The calculations assume that the facility is operating for 30 years. Table 4-8 shows the predicted concentrations of compounds in 1 cm soil in the watershed and the predicted concentrations of compounds in the surface water in the Lakes complex used to evaluate both noncancer and cancer risks in the Existing Operation - Fuel Oil #6 - High Load Scenario. Predicted concentrations in 1 cm soil in the watershed and the predicted concentrations of compounds in the surface water in the Lakes complex used to evaluate both noncancer and cancer risks at the maximum hypothetical residential location, the City of Saint John, and the Farm in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 4-9. Table 4-10 shows the predicted concentrations of compounds in 1 cm soil in the watershed and the predicted concentrations of compounds in the surface water in the Lakes complex used to evaluate both noncancer and cancer risks at the maximum hypothetical residential location, the City of Saint John, and the Farm in the Natural Gas - High Load Scenario.

4.4.4 Concentrations in Produce and Vegetation

CPCs emitted from the facility may be deposited onto soil surfaces. Deposition may also occur directly onto vegetation. Uptake by plants of compounds in the vapor state can also occur. Above-ground crops can, therefore, potentially be exposed to compounds by deposition, by root uptake from the soil, or by direct vapor uptake. Below-ground crops are assumed to be exposed to compounds via root uptake from the soil. Uptake in plants through deposition, root uptake, and vapor uptake are discussed in the following sections.

Uptake into Plants via Direct Deposition

The compound concentration due to direct deposition onto the plant is a function of the rate of deposition onto the edible surfaces of the plant, the interception fraction, the weathering half-life, the crop yield, and the time until harvest. The compound concentration due to deposition is calculated for each produce type using the following equation:

$$A = B \times C \times \frac{1 - e^{-DE}}{F \times D}$$

where:

A = Compound Concentration in Produce Type due to Deposition (mg/kg);
 B = Compound Deposition Rate (mg/m²/day);
 C = Interception Fraction of the Edible Surface of Plant (unitless);
 D = Plant Surface Loss Coefficient (1/day) (K_{el}),
 E = Length of Plant Exposure to Deposition (day); and
 F = Yield or Standing Crop Biomass (kg/m²)

where:

$K_{el} = \ln(2) \div \text{Weathering Half-life (day)}$;

Compound deposition rates were estimated from emissions using the following formula:

$$A = B \times (1 - C) \times [D + E \times F]$$

where:

A = Compound Deposition Rate (mg/m²/day)
 B = Compound Emission Rate (g/sec)
 C = Fraction of Compound Emitted in Vapor Form (unitless)
 D = Unitized Dry Deposition Rate (g/m²/y)/(g/sec)

E = Fraction of Wet Deposition Adhering to Plant Surface (unitless)
F = Unitized Wet Deposition Rate (g/m²/y)/(g/sec)

Unitized deposition rates resulting from emissions from the CCGS are presented in Table 2-7 for the Existing Operation - Fuel Oil #6 - High Load Scenario, Table 2-8 for the Refurbishment Project - Orimulsion® - High Load Scenario, and Table 2-9 for the Natural Gas - High Load Scenario. Compound emission rates resulting from emissions from the CCGS are presented in Table 2-2. Table 4-1 shows the chemical-specific parameters necessary for estimating concentrations of CPCs in all media, including the fraction of emitted compounds assumed to be in the vapor form (U.S. EPA, 1998a, 2001d). According to U.S. EPA guidance (1998a), some of the particle-bound chemicals that deposit onto plants during rain events (wet deposition) can be expected to wash off of the plant as rain drops fall off the plant and onto the soil. U.S. EPA (1998a) recommends a value of 0.6 for the fraction of wet deposition adhering to plant surfaces for organics and cations.

Produce type-specific interception factors are a measure of the fraction of the plant which is available for deposition. These factors are obtained from U.S. EPA (1998a) and are presented in Table 4-11.

The particulate weathering half-life denotes the length of time assumed for one-half of the particulates deposited onto the surface of the plant to be removed via weathering (e.g., precipitation, dew, wind). U.S. EPA (1998a) recommends a particulate weathering half-life of 14 days, corresponding to a plant surface loss coefficient of 18 yr⁻¹.

The produce-specific time until harvest is a measure of the length of time required for the plant to grow to full maturity, at which time it is assumed to be harvested. This risk assessment assumes that deposition of particulates onto the surfaces of the plant occurs every day prior to harvesting. The values for harvest time used in this risk assessment are obtained from U.S. EPA (1998a). These values are presented in Table 4-11.

The produce-specific crop yield is a measure of the density of plants grown in a given area. Produce-specific crop yields used in this risk assessment are derived from U.S. EPA (1998a) and are shown in Table 4-11.

Root Uptake

The compound concentration due to root uptake is calculated for all produce types using the following formula:

$$A = B \times C \times D$$

where:

A = Compound Concentration in Produce Type Due to Root Uptake (mg/kg);

B = Compound Concentrations in Soil (mg/kg dry weight);

C = Soil-Plant Bioconcentration Factor (kg compound in plant/kg compound in soil); and

D = Empirical Correction Factor in Produce Type (unitless).

The methodology used to derive compound concentrations in soil is discussed in Section 4.4.2. Compound concentrations in soil resulting from facility emissions are presented in Table 4-5 for the Existing Operation - Fuel Oil #6 - High Load Scenario, Table 4-6 for the Refurbishment Project - Orimulsion® - High Load Scenario, and Table 4-7 for the Natural Gas - High Load Scenario.

U.S. EPA (1998a) provides soil-plant bioconcentration factors for above-ground and below-ground crops. The soil-plant bioconcentration factors used in this risk assessment are shown in Table 4-1. U.S. EPA (1998a) recommends using an empirical correction factor of 0.01 to estimate concentrations of compounds with log K_{ow} greater than 4 in below-ground crops. For above-ground crops and for compounds with log K_{ow} less than 4 in below-ground crops, U.S. EPA (1998a) recommends an empirical correction factor of 1.

Vapor-Phase Absorption

The compound concentration in plants due to vapor transfer into plants is estimated using the following formula:

$$A = B \times C \times \frac{D \times E \times F}{G}$$

where:

A = Compound Concentration in Plant Due to Vapor Transfer (mg/kg plant)

B = Compound Emission Rate (g/sec)

C = Fraction of Compound Emitted in Vapor Form (unitless)

D = Unitized Concentration of Compound Vapor in Air ($\mu\text{g}/\text{m}^3$)/(g/sec)

E = Air-to-Plant Biotransfer Factor (mg/g plant)/(mg/g air)

F = Empirical Correction Factor (unitless)

G = Density of Air (g/m^3)

Compound emission rates due to facility emissions are presented in Table 2-2. Table 4-1 shows the fraction of each compound assumed to be emitted in vapor form (U.S. EPA, 1998a, 2001d). Unitized air concentrations are shown in Table 2-7 for the Existing Operation - Fuel Oil #6 - High Load Scenario, Table 2-8 for the Refurbishment Project - Orimulsion® - High Load Scenario, and Table 2-9 for the Natural Gas - High Load Scenario. Air-to-plant transfer factors are provided in U.S. EPA (1998a) and are shown in Table 4-1. U.S. EPA (1998a) recommends using an empirical correction factor of 0.01 for compounds with a log K_{ow} greater than 4 to estimate concentrations in above-ground plants due to uptake of vapors. To estimate concentrations of compounds with log K_{ow} less than 4, U.S. EPA (1998a) recommends an empirical correction factor of 1. No empirical correction factor is needed for below-ground crops because below-ground crops are not assumed to be exposed via uptake of vapors.

Above-ground produce, forage, and silage are assumed to be exposed to facility emissions via direct deposition, root uptake, and uptake of vapors. Grains and below-ground produce are assumed to be exposed to facility emissions via root uptake only. When the compound concentration due to these pathways have been determined for each produce type, the total compound concentration in each produce type can be estimated using the following formula:

$$A = B + C + D$$

where:

A = Total Concentration in Produce Type (mg/kg);
B = Concentration in Produce Type due to Root Uptake (mg/kg);
C = Concentration in Produce Type due to Deposition (mg/kg); and
D = Concentration in Produce Type due to Uptake of Vapors (mg/kg).

Table 4-12 shows the predicted concentrations of compounds in all produce types used to evaluate both noncancer and cancer risks at the maximum hypothetical residential location, the City of Saint John, and the Farm in the Existing Operation - Fuel Oil #6 - High Load Scenario. Predicted concentrations in all produce types used to evaluate both noncancer and cancer risks at the maximum hypothetical residential location, the City of Saint John, and the Farm in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 4-13. Table 4-14 shows the predicted concentrations of compounds in all produce types used to evaluate both noncancer and cancer risks at the maximum hypothetical residential location, the City of Saint John, and the Farm in the Natural Gas - High Load Scenario.

4.4.5 Concentrations in Fish

Compounds in surface water as a result of deposition of particles onto the Lakes complex and the watershed may be taken up into fish. Compound concentrations in fish are a function of the concentration of a compound in the water and the tendency of a compound to accumulate in fish tissue. The methodology used to estimate compound concentrations in fish is consistent with U.S. EPA (1998a, 1999b, 2000). The following equation is used to estimate compound concentrations in fish:

$$A = B \times C$$

where:

A = Compound Concentration in Fish (mg/kg)
B = Compound Concentration in Surface Water (mg/L)
C = Fish Bioconcentration Factor or Fish Bioaccumulation Factor (mg/kg fish) / (mg/L)

Compound Concentrations in Surface Water

Section 4.4.3 describes the methodology used to estimate concentrations of compounds in surface water in the Lakes complex. Table 4-8 presents the concentrations of compounds in

surface water from the Lakes complex resulting from facility emissions in the Existing Operation - Fuel Oil #6 - High Load Scenario. Table 4-9 presents the concentrations of compounds in surface water from the Lakes complex resulting from facility emissions in the Refurbishment Project - Orimulsion® - High Load Scenario. Table 4-10 presents the concentrations of compounds in surface water from the Lakes complex resulting from facility emissions in the Natural Gas - High Load Scenario.

Fish Bioconcentration Factors and Fish Bioaccumulation Factors

The fish bioconcentration factor (BCF) and the fish bioaccumulation factor (BAF) each represent the ratio of the concentration of compound in the fish tissue to the concentration of dissolved compound in the water body. This value represents the tendency for a compound to accumulate in the fish tissue after the fish has been exposed to the compound in the water body. EPA (1998a) recommends using BCFs for certain metals and organics with a log Kow less than 4. For organic compounds with log Kow greater than 4 and for other metals (lead and mercury), U.S. EPA (1998a) recommends using BAFs. BCFs and BAFs were obtained from U.S. EPA (1998a). The product of the compound concentration in the water body and the BCF or BAF provides an estimate of the compound concentration in the fish tissue. The BCFs and BAFs used in this risk assessment are shown in Table 4-1.

Table 4-8 presents the concentrations of compounds in fish from the Lakes complex used to evaluate both noncancer and cancer risks, resulting from facility emissions in the Existing Operation - Fuel Oil #6 - High Load Scenario. Table 4-9 presents the concentrations of compounds in fish from the Lakes complex used to evaluate both noncancer and cancer risks, resulting from facility emissions in the Refurbishment Project - Orimulsion® - High Load Scenario. Table 4-10 presents the concentrations of compounds in fish from the Lakes complex used to evaluate both noncancer and cancer risks, resulting from facility emissions in the Natural Gas - High Load Scenario.

4.4.6 Concentrations in Animal Products

Compounds in soil and produce as a result of deposition of particles may be taken up into animals as a result of consumption of vegetation and incidental ingestion of soil. Compound concentrations in beef, dairy, pork, poultry, and eggs are a function of the concentration of the compound in the soil, the concentration of the compound in the animal's feed, the animal's soil ingestion rate, the consumption rate of feed, and the tendency of a compound to accumulate in animal tissue. The methodology used to estimate compound concentrations in animal products is consistent with U.S. EPA (1998a, 1999b, 2000). The following equation is used to estimate compound concentrations in animal products:

$$A = (\sum (B_i \times C_i) + D \times E) \times F$$

where:

A = Compound Concentration in Animal Product (mg/kg tissue);
B = Quantity of Plant Type *i* Ingested by Animal (kg/day);
C = Compound Concentration in Plant Type *i* (mg/kg);
D = Quantity of Soil Ingested by Animal (kg/day);
E = Compound Concentration in Soil (mg/kg);
F = Biotransfer Factor for Animal Product (mg/kg animal product)/(mg/day)

This approach is consistent with U.S. EPA (1998a). In this approach, the amount of various dietary components and the amount of soil ingested by the animal is estimated. Next, the concentration of the chemical in each medium to which that animal is exposed is estimated. Then, a biotransfer factor is employed to estimate the amount of chemical in the animal product. Biotransfer factors are calculated from bioconcentration factors by taking into account the amount of diet and soil ingested by the animal.

Beef and dairy cattle are assumed to ingest forage, silage, and grain. Pigs are assumed to ingest silage and grain. Poultry are assumed to ingest grain. The compound concentration in each crop type (forage, grain, silage) is calculated using the approach described above for estimating concentrations of compounds in above-ground and below ground produce (Section 4.4.4). Concentrations of compounds in silage were estimated assuming that silage is exposed to facility emissions via direct deposition, root uptake from subsurface soil, and vapor uptake. Compound concentrations in forage are assumed to be due to direct deposition, root uptake from surface soil, and vapor uptake. Compound concentrations in grain are due to root uptake from subsurface soil. Plant-specific factors used to estimate compound concentrations in plant types are shown in Table 4-1 and were obtained from U.S. EPA (1998a).

Compound concentrations in soil resulting from facility emissions are presented in Tables 4-5 (Existing Operation - Fuel Oil #6 - High Load Scenario), 4-6 (Refurbishment Project - Orimulsion® - High Load Scenario), and 4-7 (Natural Gas - High Load Scenario). Compound concentrations in vegetation resulting from facility emissions are presented in Tables 4-12 (Existing Operation - Fuel Oil #6 - High Load Scenario), 4-13 (Refurbishment Project - Orimulsion® - High Load Scenario), and 4-14 (Natural Gas - High Load Scenario). Soil and plant ingestion rates for animals are presented in Table 4-11 and were obtained from U.S. EPA (1998a). Root uptake factors used in this risk assessment are presented in Table 4-1 and are discussed in Section 4.5.4.

The biotransfer factor (BTF) estimates the extent to which certain compounds are transferred from the feed and soil ingested by the animal to the animal tissue. The values used in this risk assessment are presented in Table 4-1 and were obtained from U.S. EPA (1998a).

Table 4-15 presents the concentrations of compounds in animal products from the farm used to evaluate both noncancer and cancer risks, resulting from facility emissions in the Existing Operation - Fuel Oil #6 - High Load Scenario. Table 4-16 presents the concentrations of compounds in animal products from the farm used to evaluate both noncancer and cancer risks, resulting from facility emissions in the Refurbishment Project - Orimulsion® - High Load Scenario. Table 4-17 presents the concentrations of compounds in animal products from the farm used to

evaluate both noncancer and cancer risks, resulting from facility emissions in the Natural Gas - High Load Scenario.

4.5 Methodology for Calculating Exposure Doses

This section describes the equations and assumptions used to evaluate receptors' potential exposures to compounds in media affected by emissions from the CCGS. The equations used to evaluate potential exposures in this risk assessment are consistent with equations presented in U.S. EPA (1998a).

The equation used to calculate Chronic Average Daily Dose (CADD) is used to estimate a receptor's potential daily intake from exposure to compounds with potential noncarcinogenic effects. According to U.S. EPA (1989), the exposure dose should be calculated by averaging over the period of time for which the receptor is assumed to be exposed. The CADD for each compound via each route of exposure is compared to the noncarcinogenic reference dose for that compound in order to estimate the potential noncarcinogenic hazard index due to exposure to that compound via that route of exposure (see Section 5.1).

For compounds with potential carcinogenic effects, however, the equation for Lifetime Average Daily Dose (LADD) is employed to estimate potential exposures. In accordance with U.S. EPA (1989), the LADD is calculated by averaging the assumed exposure over the receptor's entire lifetime (assumed to be 70 years). The LADD for each compound via each route of exposure is combined with the cancer slope factor for that compound in order to estimate the potential carcinogenic risk due to exposure to that compound via that route of exposure (see Section 5.2).

Two age groups were evaluated for each receptor scenario. The Child age group was assumed to comprise a six-year duration from ages 1-6. The Adult age group was assumed to have a duration of 30 years. Both carcinogenic and noncarcinogenic risks were estimated for each age group. Potential risks to women of child-bearing age are discussed in the uncertainty section.

The equations for estimating a receptor's average daily dose (both lifetime and chronic) are presented in the following subsections. The exposure parameters used in each potential exposure pathway are also discussed in the following subsections, and are shown in Table 4-18.

4.5.1 Estimation of Potential Exposure via Inhalation

Receptors assumed to live near the proposed facility may inhale compounds emitted from the facility in a gaseous state, in a particulate state, or bound to particulates in the air. The Chronic Average Daily Dose (CADD) and the Lifetime Average Daily Dose (LADD) calculations estimating potential inhalation exposure are presented in Appendix B. The equation used to calculate the CADD and LADD due to inhalation exposure is as follows:

$$A = \frac{B \times C \times D \times E}{F \times G}$$

where:

A = CADD or LADD following Inhalation (mg/kg-day)
B = Compound Concentration in Ambient Air (mg/m³)
C = Inhalation Rate (m³/day)
D = Exposure Frequency (days/year)
E = Exposure Duration (years)
F = Body Weight (kg)
G = Averaging Time (days)

Inhalation exposure is a function of the ambient concentration of the air concentration, inhalation rate, exposure frequency, exposure duration, and human body weight. Each of the parameters in these equations is described below.

Compound Concentration in Ambient Air

The methodology for calculating compound concentrations in ambient air is presented in Section 2.3. Predicted compound concentrations in ambient air for the various locations are shown in Tables 4-2 (Existing Operation - Fuel Oil #6 - High Load Scenario), 4-3 (Refurbishment Project - Orimulsion® - High Load Scenario), and 4-4 (Natural Gas - High Load Scenario).

Inhalation Rate

The inhalation rates assumed for the receptors in this risk assessment are presented in Table 4-18. Daily inhalation rates for children and adults were obtained from Health Canada (1994). An inhalation rate of 23 m³/day was used for the adult, and an inhalation rate of 5 m³/day was used for the child. The calculation of average daily doses of chemicals via inhalation assumes that emissions will be continuous for a 30-year operating period.

Body Weight

As shown in Table 4-18, the body weights assumed in this risk assessment are 13 kg for the child and 70 kg for the adult receptors (Health Canada, 1994).

Exposure Duration

The exposure durations are presented in Table 4-18. It was assumed that the child is exposed for six years and the adult is exposed for 30 years.

Averaging Time

The average daily dose of compounds used to calculate noncarcinogenic risks must be averaged over the duration which the receptor is assumed to be exposed (U.S. EPA, 1989). For the child receptor, this is 2,190 days (six years). For the adult receptor, the noncancer averaging time is 10,950 days (30 years).

The average daily dose used to determine potential carcinogenic effects, however, must be averaged over the entire lifetime (70 years, or 25,550 days), regardless of the length of time which the receptor is assumed to be exposed (U.S. EPA, 1989). The Averaging Time values for the receptors are presented in Table 4-18.

4.5.2 Estimation of Potential Exposure via Contact with Soil

For purposes of this study it was assumed that CPCs may be deposited onto soil surfaces within the study area. Children can incidentally ingest soil while playing outdoors, while adults may incidentally ingest soil while engaged in outdoor activities such as gardening or sports. This approach is consistent with EPA guidance on performing risk assessments for combustor facilities (U.S. EPA, 1998a).

The Chronic Average Daily Dose (CADD) and the Lifetime Average Daily Dose (LADD) calculations estimating potential soil ingestion and dermal contact exposure are presented in Appendix B. The equation used to calculate the CADD and LADD due to soil ingestion and dermal contact is as follows:

$$A = \frac{B \times C \times D \times E}{F \times G}$$

where:

- A = CADD or LADD following Incidental Ingestion of Soil (mg/kg-day);
- B = Compound Concentration in Surface Soil (mg/kg);
- C = Soil Ingestion Rate (mg/day);
- D = Exposure Frequency (days/year);
- E = Exposure Duration (years)
- F = Body Weight (kg);
- G = Averaging Time (years).

Each of the parameters used in these equations is described below.

Compound Concentration in Surface Soil

Receptors are assumed to contact soil from the top one centimeter (defined as surface soil). The methodology used to determine concentrations of compounds in soil is presented in Section 4.4.2. Surface soil concentrations at the various receptor locations resulting from emissions from the

facility are presented in Tables 4-5 (Existing Operation - Fuel Oil #6 - High Load Scenario), 4-6 (Refurbishment Project - Orimulsion® - High Load Scenario), and 4-7 (Natural Gas - High Load Scenario).

Soil Ingestion Rate

The soil ingestion rates used in this risk assessment are 200 mg/day for the child and 20 mg/day for the adult. The soil ingestion rate for the child is derived from information in U.S. EPA (1997c), while that for the Adult is derived from Health Canada (1994). Health Canada (1994) provides a lower soil ingestion rate of 50 mg/day for the child, however, the more conservative value recommended by U.S. EPA (1997c) has been used in this risk assessment. These values are presented in Table 4-18.

Exposure Frequency

The risk assessment assumes that receptors can contact soil outdoors at an exposure frequency of 350 days per year. This frequency assumes that receptors take two weeks of vacation each year. These values are presented in Table 4-18 for the various receptors.

Body Weight

As shown in Table 4-18, the body weights assumed in this risk assessment are 13 kg for the child and 70 kg for the adult receptors (Health Canada, 1994).

Exposure Duration

The exposure durations are presented in Table 4-18. It was assumed that the child is exposed for six years and the adult is exposed for 30 years.

Averaging Time

The average daily dose of compounds used to calculate noncarcinogenic risks must be averaged over the duration which the receptor is assumed to be exposed (U.S. EPA, 1989). For the child receptor, this is 2,190 days (six years). For the adult receptor, the noncancer averaging time is 10,950 days (30 years).

The average daily dose used to determine potential carcinogenic effects, however, must be averaged over the entire lifetime (70 years, or 25,550 days), regardless of the length of time which the receptor is assumed to be exposed (U.S. EPA, 1989). The Averaging Time values for the receptors are presented in Table 4-18.

4.5.3 Estimation of Potential Exposure via Ingestion of Drinking Water

Exposure to drinking water was evaluated for adult and child receptors. Receptors were assumed to ingest water from the Lakes complex. The approach used to estimate potential exposure via

drinking water ingestion is consistent with U.S. EPA guidance on performing risk assessments for combustor facilities (U.S. EPA, 1998a). The Chronic Average Daily Dose (CADD) and the Lifetime Average Daily Dose (LADD) calculations are presented in Appendix B. The equation used to calculate the CADD and LADD due to ingestion of drinking water is as follows:

$$A = \frac{B \times C \times D \times E}{F \times G}$$

where:

A = CADD or LADD following Ingestion of Drinking Water (mg/kg-day);
B = Compound Concentration in Water (mg/L);
C = Water Ingestion Rate (L/day);
D = Exposure Frequency (days/year);
E = Exposure Duration (years);
F = Body Weight (kg); and
G = Averaging Time (days)

Each of the parameters used in these equations is described below.

Compound Concentration in Water

Receptors are assumed to ingest surface water from the Lakes complex as drinking water. The methodology used to derive compound concentrations in surface water is described in Section 4.4.3. Compound concentrations in surface water from the Lakes complex resulting from facility emissions are presented in Tables 4-8 (Existing Operation - Fuel Oil #6 - High Load Scenario), 4-9 (Refurbishment Project - Orimulsion® - High Load Scenario), and 4-10 (Natural Gas - High Load Scenario).

Water Ingestion Rate

The water ingestion rates for both the child and the adult are derived from Health Canada (1994). The child receptor was assumed to ingest 0.8 L/day, and the adult receptor was assumed to ingest 1.5 L/day. These values are presented in Table 4-18 for the various receptors.

Exposure Frequency

The risk assessment assumes that receptors can ingest drinking at an exposure frequency of 350 days per year. This frequency assumes that receptors take two weeks of vacation each year. These values are presented in Table 4-18 for the various receptors.

Body Weight

As shown in Table 4-18, the body weights assumed in this risk assessment are 13 kg for the child and 70 kg for the adult receptors (Health Canada, 1994).

Exposure Duration

The exposure durations are presented in Table 4-18. It was assumed that the child is exposed for six years and the adult is exposed for 30 years.

Averaging Time

The average daily dose of compounds used to calculate noncarcinogenic risks must be averaged over the duration which the receptor is assumed to be exposed (U.S. EPA, 1989). For the child receptor, this is 2,190 days (six years). For the adult receptor, the noncancer averaging time is 10,950 days (30 years).

The average daily dose used to determine potential carcinogenic effects, however, must be averaged over the entire lifetime (70 years, or 25,550 days), regardless of the length of time which the receptor is assumed to be exposed (U.S. EPA, 1989). The Averaging Time values for the receptors are presented in Table 4-18.

4.5.4 Estimation of Potential Exposure via Consumption of Produce

Potential exposure is a function of the amount of produce ingested per day, concentration of chemicals associated with the produce, percentage of ingested produce that is home-grown, growing season, weathering removal, and human body weight. This approach is consistent with EPA guidance on performing risk assessments for combustor facilities (U.S. EPA, 1998a).

The Chronic Average Daily Dose (CADD) and the Lifetime Average Daily Dose (LADD) calculations estimating potential exposure via produce consumption are presented in Appendix B. The equation used to calculate the CADD and LADD due to produce consumption is as follows:

$$A = \frac{B \times C \times D \times E \times F}{G \times H}$$

where:

A = CADD or LADD following Ingestion of Produce (Above-ground or Below-ground) (mg/kg-day);
B = Compound Concentration in Produce (Above-ground or Below-ground) (mg/g);
C = Consumption Rate for Produce (Above-ground or Below-ground) (g/day);
D = Fraction of Produce which is Homegrown;
E = Exposure Frequency (days/year)
F = Exposure Duration (years); and
G = Body Weight (kg);
H = Averaging Time (days)

Each of the parameters used in these equations is described below.

Compound Concentration in Produce Type

Concentrations were predicted in above-ground plants by assuming root uptake of chemicals in soil, direct deposition of chemicals in the air, and vapor uptake into leaves. Concentrations in below-ground plants were predicted by assuming root uptake of chemicals in soil. Compound concentrations in produce types are shown in Tables 4-12 (Existing Operation - Fuel Oil #6 - High Load Scenario), 4-13 (Refurbishment Project - Orimulsion® - High Load Scenario), and 4-14 (Natural Gas - High Load Scenario)

Consumption Rate for Produce Type

Consumption rates for each produce type for the receptor were obtained from U.S. EPA (1998a) and are presented in Table 4-18. As shown in Table 4-19, the produce type consumption rates are identical for each of the receptors. The farmer is assumed to grow produce for personal consumption. This receptor is not assumed to grow large quantities of produce to sell commercially.

Fraction of Produce that is Homegrown

As shown in Table 4-18, except for the farmer, the 25% of the produce consumed by the receptors is assumed to originate from the receptor's backyard garden. This value was obtained from U.S. EPA (1998a). For the farmer, it was assumed that 100% of the produce consumed originates from the garden (U.S. EPA, 1998a).

Body Weight

As shown in Table 4-18, the body weights assumed in this risk assessment are 13 kg for the child and 70 kg for the adult receptors (Health Canada, 1994).

Exposure Duration

The exposure durations are presented in Table 4-18. It was assumed that the child is exposed for six years and the adult is exposed for 30 years.

Averaging Time

The average daily dose of compounds used to calculate noncarcinogenic risks must be averaged over the duration which the receptor is assumed to be exposed (U.S. EPA, 1989). For the child receptor, this is 2,190 days (six years). For the adult receptor, the noncancer averaging time is 10,950 days (30 years).

The average daily dose used to determine potential carcinogenic effects, however, must be averaged over the entire lifetime (70 years, or 25,550 days), regardless of the length of time which

the receptor is assumed to be exposed (U.S. EPA, 1989). The Averaging Time values for the receptors are presented in Table 4-18.

4.5.5 Estimation of Potential Exposure via Consumption of Fish

As described in Section 4.2, residents who live in the study area may fish in the Lakes complex. Receptors may be exposed to compounds in the Lakes complex via consumption of fish which have taken up compounds from the water. Exposure to compounds via consumption of fish which have taken up compounds from the surface water is a function of the concentration of compound in the surface water, the tendency of the compound to bioconcentrate in the fish tissue, the receptor's fish consumption rate, and the receptor's body weight. The Chronic Average Daily Dose (CADD) and the Lifetime Average Daily Dose (LADD) calculations estimating potential exposure via consumption of fish which have taken up compounds from surface water are presented in Appendix B. The equation used to calculate the CADD and LADD due to consumption of fish which have taken up compounds from surface water is as follows:

$$A = \frac{B \times C \times D \times E}{F \times G}$$

where:

A = Chronic Average Daily Dose due to Consumption of Fish (mg/kg-day);

B = Compound Concentration in Fish (mg/kg);

C = Fish Consumption Rate (kg/day);

D = Exposure Frequency (days/year)

E = Exposure Duration (years);

F = Body Weight (kg); and

G = Averaging Time (years)

Each of the parameters used in these equations is described below:

Compound Concentrations in Fish

Compounds in surface water in the Lakes complex as a result of direct deposition, runoff, and erosion may be taken up by fish. Compound concentrations in fish are discussed in Section 4.4.5 and are presented in Tables 4-8 (Existing Operation - Fuel Oil #6 - High Load Scenario), 4-9 (Refurbishment Project - Orimulsion® - High Load Scenario), and 4-10 (Natural Gas - High Load Scenario).

Fish Consumption Rate

Only recreational fishers were assumed to be exposed via consumption of fish. The fish consumption rates assumed for the receptors are shown in Table 4-18. For children, a fish consumption rate of 11.4 g/day was used, and a consumption rate of 81.9 g/day was used for adults. The values are derived from U.S. EPA (1998a). These consumption rates are high-end

consumption rates that likely overestimate actual consumption rates for freshwater recreational fishers in new Brunswick. The extent to which these fish consumption rates overestimate actual exposures is discussed in the Uncertainty Assessment.

Body Weight

As shown in Table 4-18, the body weights assumed in this risk assessment are 13 kg for the child and 70 kg for the adult receptors (Health Canada, 1994).

Exposure Duration

The exposure durations are presented in Table 4-18. It was assumed that the child is exposed for six years and the adult is exposed for 30 years.

Averaging Time

The average daily dose of compounds used to calculate noncarcinogenic risks must be averaged over the duration which the receptor is assumed to be exposed (U.S. EPA, 1989). For the child receptor, this is 2,190 days (six years). For the adult receptor, the noncancer averaging time is 10,950 days (30 years).

The average daily dose used to determine potential carcinogenic effects, however, must be averaged over the entire lifetime (70 years, or 25,550 days), regardless of the length of time which the receptor is assumed to be exposed (U.S. EPA, 1989). The Averaging Time values for the receptors are presented in Table 4-18.

4.5.6 Estimation of Potential Exposure via Consumption of Animal Products

A review of land use patterns within a 20-km radius of the facility indicates that farming activities have taken place on a peninsula northeast of the facility. A farmer living in this area may be exposed to facility emissions via consumption of animal products (beef, dairy products, pork, poultry, eggs) which have been exposed to compounds emitted from the facility. Exposure to animal products is a function of the concentration of compounds in the animal tissue, the receptor's consumption rate of that product, and the receptor's body weight.

The Chronic Average Daily Dose (CADD) and the Lifetime Average Daily Dose (LADD) calculations estimating potential exposure via consumption of animal products are presented in Appendix B. The equation used to calculate the CADD and LADD due to consumption of animal products is as follows:

$$A = \frac{B \times C \times D \times E}{F \times G}$$

where:

A = Chronic Average Daily Dose due to Consumption of Animal Product (mg/kg-day);
B = Compound Concentration in Animal Product (mg/kg);
C = Animal Product Consumption Rate (kg/day);
D = Exposure Duration (days/year);
E = Exposure Duration (years);
F = Body Weight (kg); and
G = Averaging Time (days)

The parameters used in these equations are described below.

Compound Concentration in Animal Products

Compound concentration in the animal tissue is a function of the animal's daily intake of compounds and the tendency of compounds to bioconcentrate in the animal tissue. The concentrations of compounds in animal products are described in Section 4.4.6 and are presented in Tables 4-15 (Existing Operation - Fuel Oil #6 - High Load Scenario), 4-16 (Refurbishment Project - Orimulsion® - High Load Scenario), and 4-17 (Natural Gas - High Load Scenario).

Animal Product Consumption Rates

Consumption of animal products was evaluated only for the farmer receptor. A beef consumption rate of 79.8 g/day was used for the adult farmer, and a beef consumption rate of 7.65 g/day was used for the child farmer (U.S. EPA, 1998a). A dairy consumption rate of 670 g/day was used for the adult farmer, and a dairy consumption rate of 283 g/day was used for the child farmer (Health Canada, 1994). A poultry consumption rate of 42.7 g/day was used for the adult farmer, and a poultry consumption rate of 6.4 g/day was used for the child farmer (U.S. EPA, 1998a). An egg consumption rate of 43.4 g/day was used for the adult farmer, and an egg consumption rate of 6.57 g/day was used for the child farmer (U.S. EPA, 1998a). A pork consumption rate of 37.1 g/day was used for the adult farmer, and a pork consumption rate of 5.97 g/day was used for the child farmer (U.S. EPA, 1998a). Table 4-18 shows the animal product consumption rates for the farmer adult and child.

Body Weight

As shown in Table 4-18, the body weights assumed in this risk assessment are 13 kg for the child and 70 kg for the adult receptors (Health Canada, 1994).

Exposure Duration

The exposure durations are presented in Table 4-18. It was assumed that the child is exposed for six years and the adult is exposed for 30 years.

Averaging Time

The average daily dose of compounds used to calculate noncarcinogenic risks must be averaged over the duration which the receptor is assumed to be exposed (U.S. EPA, 1989). For the child

receptor, this is 2,190 days (six years). For the adult receptor, the noncancer averaging time is 10,950 days (30 years).

The average daily dose used to determine potential carcinogenic effects, however, must be averaged over the entire lifetime (70 years, or 25,550 days), regardless of the length of time which the receptor is assumed to be exposed (U.S. EPA, 1989). The Averaging Time values for the receptors are presented in Table 4-18.

Table 4-1

Chemical Specific Parameters:
Human Health Risk Assessment
Colleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Chemical	ksg (year) ⁻¹	Fv (unitless)	Kds (cm ³ /g)	H (atm ⁻¹ m ³ /mol)	Da (cm ³ /s)	Kow (unitless)	Kdbs (L/kg)	Kdbs (cm ³ /g)	Dw (cm ³ /s)	Particle type	Br _{soil} (unitless)	Br _{air} (unitless)	Br _{org} (unitless)	RCF (μg COPC/g DW plant)/(μg COPC/mL soil water)	Bv _{soil} (unitless)
Mercury, elemental	0	1	1.00E+03	0.0071	0.0109	NA	1000	3000	0.0000301	Particle bound	NA	NA	NA	NA	NA
Mercury, methyl	0	0.85	7.00E+03	4.70E-07	0.0528	NA	100000	3000	6.11E-06	Particle bound	0.099	0.0294	0	NA	NA
Mercury, divalent	0	0.85	5.80E+04	7.1E-10	0.0453	0.61	100000	50000	0.00000525	Particle bound	0.036	0.0145	0	NA	1800
1,4-Dichlorobenzene	1.41	1	6.16E+00	0.0028	0.0414	2560	46.2	24.6	0.00000885	Particle bound	17	0.413	0.413	105	0.086
Acenaphthene	2.48	0.999975	4.90E+01	1.84E-04	4.21E-02	9.22E+03	3.87E+02	1.96E+02	7.19E-06	Particle bound	5.48	1.98E-01	1.98E-01	2.69E+02	5.07
Acenaphthylene	0.133	0.196376	6.80E+02	1.14E-08	0.0272	100000	5100	2720	0.00000714	Particle bound	2.44	0.0498	0.0498	1660	1040000
Anthracene	0.55	0.999781	2.35E+02	0.000111	0.0324	29500	1760	940	0.00000774	Particle bound	2.76	0.101	0.101	649	29
Arsenic	NA	0	2.90E+01	NA	1.07E-01	NA	29	29	0.0000124	Particle phase	0.008	0.00633	0.036	NA	NA
Benzene	3.89	1	6.20E-01	0.00549	0.117	137	4.65	2.48	0.0000102	Particle bound	26.7	2.25	2.25	16.6	0.00192
Benzo(a)anthracene	0.372	0.88052	2.60E+03	0.00000362	0.0247	477000	19500	10400	0.00000621	Particle bound	2.11	0.0202	0.0202	5480	17200
Benzo(a)pyrene	0.477	0.26462	9.69E+03	0.000000836	0.0218	1350000	72700	38700	0.00000585	Particle bound	1.26	0.0111	0.0111	12200	225000
Benzo(b)fluoranthene	0.415	0.82198	8.36E+03	0.000000618	0.0228	1590000	62700	33400	0.00000549	Particle bound	1.66	0.01007	0.01007	13900	36500
Benzo(k)fluoranthene	0.118	0.148943	8.32E+03	0.000000415	0.0228	1560000	62400	33300	0.00000549	Particle bound	1.66	0.0101	0.0101	13800	540000
Benzo(g,h,i)perylene	1.33E-01	0.196376	6.80E+02	1.14E-08	2.72E-02	1.00E+05	5.10E+03	2.72E+03	7.14E-06	Particle bound	2.44	4.98E-02	4.98E-02	1.66E+03	1.04E+06
Beryllium	NA	0	7.90E+02	NA	4.39E-01	NA	790	790	0.00000508	Particle phase	0.0015	0.00258	0.01	NA	NA
Cadmium	NA	0	7.50E+01	NA	8.16E-02	NA	75	75	0.00000945	Particle phase	0.064	0.125	0.364	NA	NA
Chromium	NA	0	1.80E+06	NA	1.01E-01	NA	1800000	1800000	0.0000463	Particle phase	0.0045	0.00488	0.0075	NA	NA
Chromium VI	NA	0	1.90E+01	NA	1.36E-01	NA	19	19	0.0000158	Particle phase	0.0045	0.00488	0.0075	NA	NA
Chrysene	0.253	0.761276	2.97E+03	0.00000121	0.0248	548000	22300	11900	0.00000621	Particle bound	2.05	0.01866	0.01866	6100	59700
Dibenz(a,h)anthracene	0.269	0.010668	1.79E+04	1.12E-08	0.018	3530000	134000	71600	0.00000601	Particle phase	0.00143	0.00636	0.00636	25600	46800000
Fluoranthene	0.575	0.992042	4.91E+02	0.00000933	0.0275	121000	3680	1960	0.00000718	Particle bound	3.9	0.0446	0.0446	1920	1560
Fluorene	4.22	0.934896	7.71E+01	9.41E-08	0.0363	14700	578	308	0.00000788	Particle bound	4.96	0.151	0.151	383	16300
Formaldehyde	36.1	1	2.62E-02	0.000278	0.5	2.2	0.196	0.105	0.0000174	Particle bound	257	24.6	24.6	6.73	0.00465
Indeno(1,2,3-cd)pyrene	0.347	0.007092	4.11E+04	4.86E-09	0.019	8220000	308000	164000	0.00000566	Particle phase	1.19	0.0039	0.0039	49100	2.67E+08
Lead	NA	0	9.00E+02	NA	0.0543	NA	900	900	0.00000628	Particle phase	0.009	0.0136	0.045	NA	NA
Naphthalene	5.27	0.999999	1.19E+01	0.000482	0.0526	2360	89.3	47.6	0.00000892	Particle bound	8.23	0.435	0.435	98.1	0.452
Nickel	NA	0	6.50E+01	NA	0.126	NA	65	65	1.46E-05	Particle phase	0.008	0.00931	0.032	NA	NA
Phenanthrene	1.26	1	5.01E+02	0.188	0.0333	35500	3760	2010	0.00000747	Particle bound	1.49	0.0908	0.0908	747	0.0208
Pyrene	0.133	0.196376	6.80E+02	1.14E-08	0.0272	100000	5100	2720	0.00000714	Particle bound	2.44	0.0498	0.0498	1660	1040000
Toluene	11.5	1	1.40E+00	0.00613	0.0972	465	10.5	5.6	0.00000923	Particle bound	23.3	1.11	1.11	32.6	0.00633
Vanadium	NA	NA	5.00E+01	NA	0.0779	NA	50	50	0.00002632	Particle phase	3.00E-03	5.50E-03	5.50E-03	NA	NA

Notes:

ksg: COPC loss constant due to biotic and abiotic degradation

Fv: Fraction of COPC air concentration in vapor phase

Kds: Soil-water partition coefficient

H: Henry's law constant

Da: Diffusivity of COPC in air

Kow: Octanol-water partition coefficient

Kdsw: Suspended sediment-surface water partition coefficient

Kdbs: Bed sediment/pore water partition coefficient

Dw: Diffusivity of COPC in water

Br_{soil}: Plant-soil bioconcentration factor for belowground produceBr_{air}: Plant-soil bioconcentration factor for aboveground produceBr_{org}: Plant-soil bioconcentration factor for forage

RCF: Root concentration factor

Bv_{soil}: Air-to-plant biotransfer factor for COPC in forageV/G_{soil}: Empirical correction factor for belowground produceV/G_{soil}: Empirical correction factor for aboveground produce (forage and silage)Ba_{beef}: Biotransfer factor for beefBa_{milk}: Biotransfer factor for milkBa_{pork}: Biotransfer factor for porkBa_{eggs}: Biotransfer factor for chicken eggsBCF_{fish}: Bioconcentration factor for fish [mg COPC/kg fish]/[mg COPC/kg dissolved water]BAF_{fish}: Bioaccumulation factor for COPC in fish (L/kg FW tissue)Ba_{chicken}: Biotransfer factor for chicken

NA: Not available

Source for all chemicals except Vanadium: US EPA, 1998a.

Vanadium source: Research Triangle Institute, 1998.

Table 4.1
Chemical Specific Parameters
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Chemical	B _{bioacc} (unitless)	B _{air} (day/kg FW tissue)	B _{soil} (day/kg FW tissue)	B _{veg} (day/kg FW tissue)	B _{fish} (day/kg FW tissue)	BCF _{fish} (unitless)	BAF _{fish} (L/kg FW tissue)
Mercury, elemental	NA	0.00078	0.000338	NA	0.00358	NA	6800000
Mercury, methyl	NA	0.00522	0.00226	3.39E-05	0.0239	NA	NA
Mercury, divalent	1800	0.00522	0.00226	3.39E-05	0.0239	NA	NA
1,4-Dichlorobenzene	0.086	6.49E-05	2.05E-05	7.86E-05	0.0269	284	NA
Acenaphthene	5.07	2.31E-04	7.32E-05	2.80E-04	7.32E-02	183E-04	607
Acenaphthylene	1040000	0.00252	0.000798	0.00306	0.00798	NA	11900
Anthracene	29	0.000741	0.000234	0.000898	0.00234	NA	2600
Arsenic	NA	0.002	0.00006	NA	NA	20	NA
Benzene	0.00192	3.44E-06	1.09E-06	4.17E-06	0.00109	24.8	NA
Benzo(a)anthracene	17200	0.012	0.00379	0.0145	0.00946	NA	5100
Benzo(a)pyrene	225000	0.0338	0.0107	0.041	0.0267	NA	9950
Benzo(b)fluoranthene	36500	0.04	0.0127	0.0484	0.0316	NA	9950
Benzo(k)fluoranthene	540000	0.0398	0.0126	0.0482	0.0314	NA	9950
Benzo(g,h,i)perylene	1.04E+06	2.52E-03	7.98E-04	3.06E-03	1.99E-03	NA	11900
Beryllium	NA	0.001	9E-07	NA	NA	62	NA
Cadmium	NA	0.00012	6.5E-06	0.000191	0.0025	250	NA
Chromium, VI	NA	0.0055	0.0015	NA	NA	190	NA
Chromium, III	NA	0.0055	0.0015	NA	NA	3	NA
Chrysene	59700	0.01377	0.004355	0.0167	0.0109	NA	6030
Dibenz(a,h)anthracene	46800000	0.0886	0.028	0.107	0.07	NA	12800
Fluoranthene	1560	0.00305	0.000665	0.00369	0.00241	NA	15700
Fluorene	16300	0.00037	0.000117	0.000448	0.117	NA	1200
Formaldehyde	0.000465	5.53E-08	1.75E-08	6.69E-08	1.75E-05	1.07	NA
Indeno(1,2,3-cd)pyrene	2.67E+08	0.207	0.0653	0.25	65.3	NA	13100
Lead	NA	0.0003	0.00025	0.00036	NA	8	NA
Naphthalene	0.452	5.92E-05	1.87E-05	7.16E-05	0.0187	215	NA
Nickel	NA	0.006	0.001	NA	NA	78	NA
Phenanthrene	0.0208	0.000892	0.000282	0.00108	0.282	NA	3300
Pyrene	1040000	0.00252	0.000798	0.00306	0.798	NA	11900
Toluene	0.00633	1.17E-05	3.69E-06	1.41E-05	0.00369	62.7	NA
Vanadium	NA	0.0025	2.00E-03	NA	NA	NA	10

Notes
kg COPC loss constant kg COPC loss constant due to biotic and abiotic degradation
F_v Fraction of COPC air; F_v Fraction of COPC air concentration in vapor phase
K_{ds} Soil-water partition cK_{ds} Soil-water partition coefficient
H Henry's law constant H Henry's law constant
D_a Diffusivity of COPC in Da Diffusivity of COPC in air
K_{ow} Octanol water partition coefficient
K_{ds} Suspended sediment K_{ds} Suspended sediment surface water partition coefficient
K_{bs} Bed sediment/pore K_{bs} Bed sediment/pore water partition coefficient
D_w Diffusivity of COPC in Dw Diffusivity of COPC in water
Br_{veg} Plant-soil bioconcentration Br_{veg} Plant-soil bioconcentration factor for belowground produce
Br_{veg} Plant-soil bioconcentration Br_{veg} Plant-soil bioconcentration factor for aboveground produce
Br_{veg} Plant-soil bioconcentration Br_{veg} Plant-soil bioconcentration factor for forage
RCF Root concentration RCF Root concentration factor

BAF_{veg} Air-to-plant bioconcentration factor for COPC in forage
VG_{veg} Empirical correction factor for belowground produce
VG_{veg} Empirical correction factor for aboveground produce (forage and silage)
BA_{veg} Bioconcentration factor for beef
BA_{veg} Bioconcentration factor for milk
BA_{veg} Bioconcentration factor for pork
BA_{veg} Bioconcentration factor for chicken eggs
BCF_{fish} Bioconcentration factor for fish [mg COPC/kg fish]/[mg COPC/kg dissolved water]
BAF_{fish} Bioaccumulation factor for COPC in fish (L/kg FW tissue)
BA_{veg} Bioconcentration factor for chicken
NA Not available
Source for all chemicals except Vanadium US EPA 1998a
Vanadium source Research Triangle Institute 1998

Table 4-2

Exposure Point Concentrations in Air Under Fuel Oil High Load Conditions
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	Concentration (ug/m3)		
	Maximum Hypothetical Residence	Saint John	Northeast Farm
Mercury, elemental	9.66E-12	4.35E-10	2.15E-10
Mercury, methyl	NC	NC	NC
Mercury, divalent	2.32E-09	1.04E-07	5.12E-08
1,4-Dichlorobenzene	5.49E-09	2.47E-07	1.22E-07
Acenaphthene	1.62E-09	7.29E-08	3.60E-08
Acenaphthylene	1.94E-11	8.42E-10	4.17E-10
Anthracene	9.36E-11	4.21E-09	2.08E-09
Arsenic	1.21E-08	4.36E-07	2.18E-07
Benzene	1.82E-08	8.19E-07	4.04E-07
Benzo(a)anthracene	3.08E-10	1.38E-08	6.81E-09
Benzo(a)pyrene	8.45E-11	3.68E-09	1.82E-09
Benzo(b)fluoranthene	2.35E-10	1.05E-08	5.19E-09
Benzo(k)fluoranthene	2.35E-10	1.02E-08	5.04E-09
Benzo(g,h,i)perylene	1.73E-10	7.51E-09	3.72E-09
Beryllium	4.40E-10	1.58E-08	7.92E-09
Cadmium	3.30E-10	1.19E-08	5.94E-09
Chromium	1.98E-08	7.13E-07	3.56E-07
Chromium VI	5.74E-09	2.07E-07	1.03E-07
Chrysene	1.83E-10	8.15E-09	4.03E-09
Dibenzo(a,h)anthracene	1.28E-10	4.62E-09	2.31E-09
Fluoranthene	3.71E-10	1.67E-08	8.24E-09
Fluorene	3.43E-10	1.54E-08	7.60E-09
Formaldehyde	2.81E-06	1.26E-04	6.24E-05
Indeno(1,2,3-cd)pyrene	1.64E-10	5.91E-09	2.96E-09
Lead	1.95E-07	7.02E-06	3.51E-06
Napthalene	8.67E-08	3.90E-06	1.93E-06
Nickel	5.72E-06	2.06E-04	1.03E-04
Phenanthrene	8.05E-10	3.62E-08	1.79E-08
Pyrene	3.26E-10	1.41E-08	7.01E-09
Toluene	5.28E-07	2.38E-05	1.17E-05
Vanadium	3.76E-05	1.35E-03	6.77E-04

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-3

Exposure Point Concentrations in Air Under Orimulsion High Load Conditions
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	Concentration (ug/m3)		
	Maximum Hypothetical Residence	Saint John	Northeast Farm
Mercury, elemental	1.42E-12	1.72E-10	8.33E-11
Mercury, methyl	NC	NC	NC
Mercury, divalent	3.41E-10	4.10E-08	1.99E-08
1,4-Dichlorobenzene	1.81E-09	2.19E-07	1.06E-07
Acenaphthene	4.96E-10	6.00E-08	2.91E-08
Acenaphthylene	5.95E-12	6.96E-10	3.40E-10
Anthracene	2.87E-11	3.47E-09	1.68E-09
Arsenic	8.73E-10	8.43E-08	4.12E-08
Benzene	2.13E-08	2.58E-06	1.25E-06
Benzo(a)anthracene	9.43E-11	1.13E-08	5.51E-09
Benzo(a)pyrene	2.79E-11	3.27E-09	1.60E-09
Benzo(b)fluoranthene	7.76E-11	9.32E-09	4.52E-09
Benzo(k)fluoranthene	7.76E-11	9.06E-09	4.42E-09
Benzo(g,h,i)perylene	5.32E-11	6.22E-09	3.04E-09
Beryllium	5.02E-11	4.85E-09	2.37E-09
Cadmium	2.40E-11	2.32E-09	1.13E-09
Chromium	2.07E-09	2.00E-07	9.78E-08
Chromium VI	6.01E-10	5.81E-08	2.84E-08
Chrysene	5.60E-11	6.71E-09	3.26E-09
Dibenzo(a,h)anthracene	3.93E-11	3.81E-09	1.86E-09
Fluoranthene	1.14E-10	1.38E-08	6.69E-09
Fluorene	1.05E-10	1.27E-08	6.15E-09
Formaldehyde	6.07E-08	7.34E-06	3.56E-06
Indeno(1,2,3-cd)pyrene	5.04E-11	4.88E-09	2.38E-09
Lead	7.42E-11	7.17E-09	3.50E-09
Napthalene	9.05E-09	1.09E-06	5.31E-07
Nickel	7.90E-07	7.63E-05	3.73E-05
Phenanthrene	2.47E-10	2.99E-08	1.45E-08
Pyrene	1.00E-10	1.17E-08	5.71E-09
Toluene	2.71E-07	3.28E-05	1.59E-05
Vanadium	3.43E-06	3.31E-04	1.62E-04

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-4

Exposure Point Concentrations in Air Under Conditions Under Natural Gas High Load Conditions
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	Concentration (ug/m3)		
	Maximum Hypothetical Residence	Saint John	Northeast Farm
Mercury, elemental	2.68E-14	1.21E-12	5.96E-13
Mercury, methyl	NC	NC	NC
Mercury, divalent	6.43E-12	2.88E-10	1.42E-10
1,4-Dichlorobenzene	1.59E-08	7.16E-07	3.53E-07
Acenaphthene	2.38E-11	1.07E-09	5.29E-10
Acenaphthylene	2.38E-11	1.03E-09	5.12E-10
Anthracene	3.17E-11	1.43E-09	7.04E-10
Arsenic	NA	NA	NA
Benzene	2.77E-08	1.25E-06	6.16E-07
Benzo(a)anthracene	2.38E-11	1.07E-09	5.26E-10
Benzo(a)pyrene	1.59E-11	6.92E-10	3.43E-10
Benzo(b)fluoranthene	2.38E-11	1.06E-09	5.25E-10
Benzo(k)fluoranthene	2.38E-11	1.03E-09	5.11E-10
Benzo(g,h,i)perylene	1.59E-11	6.90E-10	3.42E-10
Beryllium	NA	NA	NA
Cadmium	NA	NA	NA
Chromium	NA	NA	NA
Chromium VI	NA	NA	NA
Chrysene	2.38E-11	1.06E-09	5.24E-10
Dibenzo(a,h)anthracene	1.59E-11	5.74E-10	2.87E-10
Fluoranthene	3.96E-11	1.78E-09	8.80E-10
Fluorene	3.70E-11	1.66E-09	8.20E-10
Formaldehyde	9.91E-07	4.46E-05	2.20E-05
Indeno(1,2,3-cd)pyrene	2.38E-11	8.58E-10	4.29E-10
Lead	NA	NA	NA
Napthalene	8.06E-09	3.63E-07	1.79E-07
Nickel	NA	NA	NA
Phenanthrene	2.25E-10	1.01E-08	5.00E-09
Pyrene	6.61E-11	2.87E-09	1.42E-09
Toluene	4.49E-08	2.02E-06	9.98E-07
Vanadium	NA	NA	NA

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 4-5
Exposure Point Concentrations in Soil Under Fuel Oil High Load Conditions
Human Health Risk Assessment
Coleston Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Concentrations in 0-1 cm Soil (mg/kg)					Concentrations in 0-20 cm Soil (mg/kg)				
	For Evaluating Cancer Risk			For Evaluating Non-Cancer Effects		For Evaluating Cancer Risk			For Evaluating Non-Cancer Effects	
	Maximum Hypothetical Residence	Saint John	Northest Farm	Maximum Hypothetical Residence	NC	Maximum Hypothetical Residence	Saint John	Northest Farm	Maximum Hypothetical Residence	NC
Mercury, elemental	1.40E-04	4.43E-06	NC	2.67E-04	NC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Mercury, methyl	7.52E-03	2.38E-04	1.36E-04	1.50E-02	4.73E-04	7.73E-06	2.44E-07	1.40E-07	1.54E-05	4.87E-07
Mercury, divalent	3.12E-07	1.04E-08	5.93E-09	3.12E-07	1.04E-08	3.80E-04	1.20E-05	6.89E-06	7.61E-04	2.40E-05
1,4-Dichlorobenzene	9.83E-06	3.27E-07	1.87E-07	9.84E-06	3.28E-07	5.21E-06	1.74E-07	9.90E-08	5.22E-06	1.74E-07
Acenaphthene	1.53E-05	3.64E-07	2.14E-07	1.78E-05	4.24E-07	6.85E-06	2.28E-07	1.30E-07	6.93E-06	2.31E-07
Acenaphthylene	5.16E-06	1.72E-07	9.80E-08	1.78E-05	4.24E-07	1.15E-06	2.74E-08	1.61E-08	1.49E-06	3.53E-08
Anthracene	2.76E-03	4.42E-05	2.44E-05	5.20E-06	1.73E-07	1.73E-06	5.78E-08	3.30E-08	1.84E-06	6.13E-08
Arsenic	1.88E-08	6.27E-10	3.58E-10	2.80E-03	4.48E-05	2.03E-03	3.24E-05	1.79E-05	2.72E-03	4.35E-05
Benzene	1.56E-04	4.98E-06	2.85E-06	1.88E-08	6.27E-10	3.71E-07	1.24E-08	7.05E-09	3.71E-07	1.24E-08
Benzo(a)anthracene	3.50E-05	8.62E-07	5.08E-07	1.70E-04	5.43E-06	8.41E-06	2.69E-07	1.54E-07	9.23E-06	2.95E-07
Benzo(a)pyrene	1.13E-04	3.54E-06	2.03E-06	3.76E-05	9.26E-07	1.78E-06	4.37E-08	2.57E-08	1.91E-06	4.70E-08
Benzo(b)fluoranthene	2.95E-04	6.85E-06	4.05E-06	1.23E-04	3.84E-06	5.80E-06	1.81E-07	1.04E-07	6.31E-06	1.97E-07
Benzo(k)fluoranthene	1.37E-04	3.25E-06	1.91E-06	3.89E-04	9.02E-06	1.54E-05	3.58E-07	2.11E-07	2.06E-05	4.79E-07
Benzo(g,h,i)perylene	1.78E-03	2.84E-05	1.57E-05	1.59E-04	3.78E-06	1.03E-05	2.44E-07	1.44E-07	1.32E-05	3.15E-07
Beryllium	1.90E-04	3.04E-06	1.67E-06	2.56E-03	4.09E-05	1.71E-04	2.74E-06	1.51E-06	3.35E-04	5.37E-06
Cadmium	1.61E-01	2.57E-03	1.42E-03	1.97E-04	3.15E-06	8.94E-05	1.43E-06	7.88E-07	1.47E-04	2.34E-06
Chromium	8.65E-04	1.38E-05	7.63E-06	3.22E-01	5.15E-03	8.05E-03	1.29E-04	7.09E-05	1.61E-02	2.57E-04
Chromium VI	1.29E-04	3.93E-06	2.28E-06	7.11E-04	1.40E-05	7.11E-04	1.14E-05	6.27E-06	8.69E-04	1.39E-05
Chrysene	2.22E-04	3.56E-06	1.97E-06	6.95E-06	2.12E-07	6.95E-06	2.12E-07	1.22E-07	8.00E-06	2.44E-07
Dibenzo(a,h)anthracene	9.74E-05	3.23E-06	1.85E-06	1.12E-05	1.80E-07	1.12E-05	1.80E-07	9.94E-08	1.28E-05	2.06E-07
Fluoranthene	1.50E-05	4.87E-07	2.79E-07	6.77E-06	2.25E-07	6.77E-06	2.25E-07	1.28E-07	7.18E-06	2.38E-07
Fluorene	5.68E-07	1.89E-08	1.08E-08	8.96E-07	2.92E-08	8.96E-07	2.92E-08	1.67E-08	9.03E-07	2.94E-08
Formaldehyde	2.30E-04	3.68E-06	2.03E-06	1.11E-05	3.71E-07	1.11E-05	3.71E-07	2.12E-07	1.11E-05	3.71E-07
Indeno(1,2,3-cd)pyrene	8.45E-01	1.35E-02	7.45E-03	1.15E-05	4.91E-07	1.15E-05	4.91E-07	1.02E-07	1.27E-05	2.05E-07
Lead	4.26E-05	1.42E-06	8.09E-07	1.25E+00	2.00E-02	7.63E-02	1.22E-03	6.73E-04	1.50E-01	2.40E-03
Naphthalene	2.87E+00	4.59E-02	2.53E-02	4.26E-05	1.42E-06	1.46E-04	4.85E-06	2.77E-06	1.46E-04	4.88E-06
Nickel	6.90E-08	2.30E-09	1.31E-09	2.96E+00	4.74E-02	1.47E+00	2.35E-02	1.29E-02	2.35E+00	3.75E-02
Phenanthrene	2.57E-04	6.12E-06	3.60E-06	6.90E-08	2.30E-09	1.15E-06	3.84E-08	2.19E-08	1.18E-06	3.86E-08
Pyrene	1.33E-06	4.43E-08	2.53E-08	3.00E-04	7.13E-06	1.93E-05	4.60E-07	2.71E-07	2.50E-05	5.93E-07
Toluene	1.46E+01	2.34E-01	1.29E-01	1.33E-06	4.43E-08	2.51E-05	8.35E-07	4.76E-07	2.51E-05	8.35E-07
Vanadium				1.50E+01	2.40E-01	8.59E+00	1.37E-01	7.58E-02	1.30E+01	2.09E-01

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-8
Exposure Point Concentrations in Soil Under Orimulsion High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Concentrations in 0-1 cm Soil (mg/kg)					Concentrations in 0-20 cm Soil (mg/kg)				
	For Evaluating Cancer Risk		For Evaluating Non-Cancer Effects			For Evaluating Cancer Risk		For Evaluating Non-Cancer Effects		
	Maximum Hypothetical Residence	NC	Saint John	Northeast Farm	NC	Maximum Hypothetical Residence	NC	Saint John	Northeast Farm	NC
Mercury, elemental	2.62E-05	1.04E-06	5.60E-07	NC	1.97E-06	1.44E-06	5.72E-08	0.00E+00	0.00E+00	0.00E+00
Mercury, methyl	1.40E-03	5.57E-05	3.00E-05	NC	1.11E-04	7.10E-05	2.82E-06	1.52E-06	1.42E-04	5.63E-06
Mercury, divalent	1.31E-07	5.61E-09	3.01E-09	NC	5.61E-09	2.18E-06	9.38E-08	5.03E-08	2.19E-06	9.40E-08
1,4-Dichlorobenzene	3.82E-06	1.64E-07	8.82E-08	NC	1.64E-07	2.66E-06	1.14E-07	6.14E-08	2.70E-06	1.16E-07
Acenaphthene	5.98E-06	1.46E-07	8.09E-08	NC	1.70E-07	4.50E-07	1.10E-08	5.08E-09	5.81E-07	1.41E-08
Acenaphthylene	2.01E-06	8.63E-08	4.63E-08	NC	8.70E-08	6.75E-07	2.90E-08	1.56E-08	7.17E-07	3.08E-08
Anthracene	2.56E-04	4.36E-06	2.22E-06	NC	4.42E-06	1.88E-04	3.20E-06	1.63E-06	2.52E-04	4.29E-06
Arsenic	2.80E-08	1.20E-09	6.46E-10	NC	2.80E-08	5.51E-07	2.37E-08	1.27E-08	5.51E-07	2.37E-08
Benzene	6.07E-05	2.45E-06	1.32E-06	NC	6.07E-05	3.27E-06	1.32E-07	7.10E-08	3.59E-06	1.45E-07
Benzo(a)anthracene	1.47E-05	3.83E-07	2.12E-07	NC	1.58E-05	2.72E-07	1.94E-08	1.07E-08	8.03E-07	2.09E-08
Benzo(a)pyrene	4.75E-05	1.85E-06	9.99E-07	NC	5.15E-05	2.43E-06	9.50E-08	5.12E-08	2.65E-06	1.03E-07
Benzo(b)fluoranthene	1.24E-04	2.89E-06	1.61E-06	NC	1.64E-04	6.50E-06	1.51E-07	8.41E-08	8.69E-06	2.02E-07
Benzo(k)fluoranthene	5.35E-05	1.30E-06	7.24E-07	NC	6.24E-05	4.02E-06	9.79E-08	5.44E-08	5.19E-06	1.26E-07
Benzo(g,h,i)perylene	2.60E-04	4.43E-06	2.25E-06	NC	3.75E-04	2.51E-05	4.27E-07	2.17E-07	4.92E-05	8.37E-07
Beryllium	1.77E-05	3.02E-07	1.54E-07	NC	1.84E-05	8.36E-06	1.42E-07	7.23E-08	1.37E-05	2.33E-07
Cadmium	2.16E-02	3.68E-04	1.87E-04	NC	4.32E-02	1.08E-03	1.84E-05	9.35E-06	2.16E-03	3.68E-05
Chromium	1.16E-04	1.98E-06	1.01E-06	NC	1.18E-04	9.56E-05	1.63E-06	8.27E-07	1.17E-04	1.99E-06
Chromium VI	5.01E-05	1.88E-06	1.02E-06	NC	5.69E-05	2.70E-06	1.02E-07	5.50E-08	3.11E-06	1.17E-07
Chrysene	8.75E-05	1.50E-06	7.62E-07	NC	9.96E-05	4.42E-06	7.58E-08	3.86E-08	5.05E-06	8.65E-08
Dibenzo(a,h)anthracene	3.80E-05	1.63E-06	8.73E-07	NC	3.96E-05	2.64E-06	1.13E-07	6.07E-08	2.80E-06	1.20E-07
Fluoranthene	5.82E-06	2.42E-07	1.30E-07	NC	5.86E-06	3.48E-07	1.45E-08	7.78E-09	3.51E-07	1.46E-08
Fluorene	1.56E-08	6.69E-10	3.59E-10	NC	1.56E-08	3.05E-07	1.31E-08	7.04E-09	3.05E-07	1.31E-08
Formaldehyde	9.06E-05	1.55E-06	7.88E-07	NC	1.00E-04	4.55E-06	7.78E-08	3.96E-08	5.03E-06	8.60E-08
Indeno(1,2,3-cd)pyrene	4.13E-04	7.03E-06	3.57E-06	NC	6.12E-04	3.73E-05	6.35E-07	3.23E-07	7.33E-05	1.25E-06
Lead	5.64E-06	2.43E-07	1.30E-07	NC	5.64E-06	1.93E-05	8.30E-07	4.46E-07	1.94E-05	8.34E-07
Naphthalene	5.09E-01	8.66E-03	4.40E-03	NC	5.26E-01	2.60E-01	4.43E-03	2.25E-03	4.16E-01	7.08E-03
Nickel	2.69E-08	1.16E-09	6.21E-10	NC	2.69E-08	4.49E-07	1.93E-08	1.04E-08	4.51E-07	1.94E-08
Phenanthrene	1.01E-04	2.45E-06	1.36E-06	NC	1.17E-04	7.56E-06	1.84E-07	1.02E-07	9.76E-06	2.38E-07
Pyrene	8.66E-07	3.72E-08	2.00E-08	NC	8.66E-07	1.63E-05	7.02E-07	3.77E-07	1.63E-05	7.02E-07
Toluene	1.71E+00	2.92E-02	1.48E-02	NC	1.76E+00	1.01E+00	1.71E-02	8.71E-03	1.53E+00	2.60E-02
Vanadium										

Notes:
NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-7

Exposure Point Concentrations in Soil Under Natural Gas High Load Conditions
Human Health Risk Assessment
Coleston Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Concentrations in 0-1 cm Soil (mg/kg)					Concentrations in 0-20 cm Soil (mg/kg)				
	For Evaluating Cancer Risk		For Evaluating Non-Cancer Effects			For Evaluating Cancer Risk		For Evaluating Non-Cancer Effects		
	Maximum Hypothetical Residence	Saint John	Northeast Farm	Maximum Hypothetical Residence	Saint John	Northeast Farm	Maximum Hypothetical Residence	Saint John	Northeast Farm	Maximum Hypothetical Residence
Mercury, elemental	NC	NC	NC	NC	NC	NC	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Mercury, methyl	3.89E-07	1.23E-08	7.05E-09	7.40E-07	2.34E-08	1.34E-08	2.14E-08	6.78E-10	3.88E-10	4.28E-08
Mercury, divalent	2.09E-05	6.59E-07	3.78E-07	4.15E-05	1.31E-06	7.52E-07	1.06E-06	3.33E-08	1.91E-08	2.11E-06
1,4-Dichlorobenzene	9.03E-07	3.01E-08	1.72E-08	9.03E-07	3.01E-08	1.72E-08	1.51E-05	5.03E-07	2.87E-07	1.51E-05
Acenaphthene	1.44E-07	4.81E-09	2.75E-09	1.45E-07	4.82E-09	2.75E-09	1.01E-07	3.35E-09	1.91E-09	1.02E-07
Acenaphthylene	1.88E-05	4.47E-07	2.63E-07	2.19E-05	5.20E-07	3.07E-07	1.41E-06	3.36E-08	1.98E-08	1.82E-06
Anthracene	1.75E-06	5.82E-08	3.32E-08	1.76E-06	5.86E-08	3.35E-08	5.87E-07	1.96E-08	1.12E-08	6.24E-07
Arsenic	NA	NA	NA	NA	NA	NA	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Benzene	2.87E-08	9.54E-10	5.45E-10	2.87E-08	9.54E-10	5.45E-10	5.65E-07	1.88E-08	1.07E-08	5.65E-07
Benzo(a)anthracene	1.21E-05	3.85E-07	2.21E-07	1.31E-05	4.20E-07	2.40E-07	6.50E-07	2.08E-08	1.19E-08	7.13E-07
Benzo(a)pyrene	6.59E-06	1.62E-07	9.52E-08	7.08E-06	1.74E-07	1.02E-07	3.34E-07	8.23E-09	4.83E-09	3.34E-07
Benzo(b)fluoranthene	1.15E-05	3.59E-07	2.08E-07	1.24E-05	3.89E-07	2.23E-07	5.88E-07	1.84E-08	1.05E-08	6.39E-07
Benzo(k)fluoranthene	2.99E-05	6.94E-07	4.10E-07	3.94E-05	9.14E-07	5.40E-07	1.56E-06	3.63E-08	2.14E-08	2.09E-06
Benzo(g,h,i)perylene	1.25E-05	2.98E-07	1.76E-07	1.46E-05	3.48E-07	2.05E-07	9.43E-07	2.24E-08	1.32E-08	1.22E-06
Beryllium	NA	NA	NA	NA	NA	NA	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium	NA	NA	NA	NA	NA	NA	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chromium	NA	NA	NA	NA	NA	NA	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chromium VI	NA	NA	NA	NA	NA	NA	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chrysene	1.67E-05	5.12E-07	2.94E-07	1.90E-05	5.81E-07	3.34E-07	9.04E-07	2.76E-08	1.59E-08	1.04E-06
Dibenz(a,h)anthracene	2.75E-05	4.43E-07	2.44E-07	3.14E-05	5.04E-07	2.78E-07	1.39E-06	2.24E-08	1.23E-08	1.59E-06
Fluoranthene	1.04E-05	3.45E-07	1.97E-07	1.08E-05	3.60E-07	2.05E-07	7.22E-07	2.40E-08	1.37E-08	7.66E-07
Fluorene	1.61E-06	5.26E-08	3.01E-08	1.62E-06	5.29E-08	3.03E-08	9.66E-08	3.15E-09	1.80E-09	9.74E-08
Formaldehyde	2.00E-07	6.67E-09	3.80E-09	2.00E-07	6.67E-09	3.80E-09	3.92E-06	1.31E-07	7.46E-08	3.92E-06
Indeno(1,2,3-cd)pyrene	3.33E-05	5.35E-07	2.95E-07	3.68E-05	5.91E-07	3.28E-07	1.67E-06	2.68E-08	1.48E-08	1.85E-06
Lead	NA	NA	NA	NA	NA	NA	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Naphthalene	3.96E-06	1.32E-07	7.52E-08	3.96E-06	1.32E-07	7.53E-08	1.35E-05	4.51E-07	2.57E-07	1.36E-05
Nickel	NA	NA	NA	NA	NA	NA	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Phenanthrene	1.93E-08	6.43E-10	3.67E-10	1.93E-08	6.43E-10	3.67E-10	3.22E-07	1.07E-08	6.13E-09	0.00E+00
Pyrene	5.22E-05	1.24E-06	7.31E-07	6.08E-05	1.45E-06	8.52E-07	3.92E-06	9.32E-08	5.49E-08	5.06E-06
Toluene	1.13E-07	3.76E-09	2.15E-09	1.13E-07	3.76E-09	2.15E-09	2.13E-06	7.10E-08	4.05E-08	2.13E-06
Vanadium	NA	NA	NA	NA	NA	NA	0.00E+00	0.00E+00	0.00E+00	0.00E+00

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 4-8

Exposure Point Concentrations in Spruce, Ludgate, and Menzies Lakes and Watershed Under Fuel Oil High Load Conditions
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	For Evaluating Cancer Risk			For Evaluating Non-Cancer Effects		
	Water (mg/L)	Watershed Soil (0-1cm) (mg/kg)	Fish (mg/kg)	Water (mg/L)	Watershed Soil (0-1cm) (mg/kg)	Fish (mg/kg)
Mercury, elemental	NC	0.00E+00	NC	NC	0.00E+00	NC
Mercury, methyl	3.06E-09	6.58E-06	2.08E-02	4.74E-09	1.25E-05	3.23E-02
Mercury, divalent	1.26E-08	3.53E-04	NC	1.87E-08	7.01E-04	NC
1,4-Dichlorobenzene	3.08E-09	1.50E-08	8.75E-07	3.08E-09	1.50E-08	8.75E-07
Acenaphthene	1.27E-09	4.72E-07	7.70E-07	1.27E-09	4.72E-07	7.70E-07
Acenaphthylene	1.29E-09	6.40E-07	1.54E-05	1.44E-09	7.46E-07	1.71E-05
Anthracene	8.57E-11	2.47E-07	2.23E-07	8.59E-11	2.49E-07	2.23E-07
Arsenic	4.11E-06	8.33E-05	8.22E-05	4.16E-06	8.45E-05	8.32E-05
Benzene	9.18E-09	9.03E-10	2.28E-07	9.18E-09	9.03E-10	2.28E-07
Benzo(a)anthracene	1.38E-09	7.35E-06	7.04E-06	1.43E-09	8.01E-06	7.29E-06
Benzo(a)pyrene	3.19E-10	1.48E-06	3.18E-06	3.26E-10	1.59E-06	3.24E-06
Benzo(b)fluoranthene	5.44E-10	5.28E-06	5.41E-06	5.59E-10	5.73E-06	5.56E-06
Benzo(k)fluoranthene	1.74E-09	1.22E-05	1.73E-05	2.05E-09	1.61E-05	2.04E-05
Benzo(g,h,i)perylene	1.15E-08	5.71E-06	1.37E-04	1.28E-08	6.65E-06	1.52E-04
Beryllium	1.23E-07	5.36E-05	7.63E-06	1.66E-07	7.71E-05	1.03E-05
Cadmium	1.11E-07	5.73E-06	2.78E-05	1.15E-07	5.94E-06	2.87E-05
Chromium	1.11E-08	4.85E-03	2.11E-06	1.91E-08	9.70E-03	3.62E-06
Chromium VI	1.95E-06	2.61E-05	5.86E-06	1.97E-06	2.63E-05	5.90E-06
Chrysene	1.63E-09	5.95E-06	9.85E-06	1.74E-09	6.76E-06	1.05E-05
Dibenzo(a,h)anthracene	7.35E-10	6.70E-06	9.41E-06	7.74E-10	7.63E-06	9.90E-06
Fluoranthene	1.12E-09	4.67E-06	1.76E-05	1.14E-09	4.86E-06	1.79E-05
Fluorene	1.61E-08	7.11E-07	1.93E-05	1.62E-08	7.16E-07	1.94E-05
Formaldehyde	1.01E-06	2.72E-08	1.08E-06	1.01E-06	2.72E-08	1.08E-06
Indeno(1,2,3-cd)pyrene	3.88E-10	6.93E-06	5.09E-06	4.01E-10	7.66E-06	5.26E-06
Lead	5.27E-05	2.55E-02	4.21E-04	7.26E-05	3.77E-02	5.81E-04
Napthalene	5.14E-08	2.04E-06	1.10E-05	5.14E-08	2.04E-06	1.10E-05
Nickel	1.93E-03	8.65E-02	1.51E-01	1.98E-03	8.93E-02	1.55E-01
Phenanthrene	4.94E-10	3.31E-09	1.63E-06	4.94E-10	3.31E-09	1.63E-06
Pyrene	2.17E-08	1.08E-05	2.58E-04	2.41E-08	1.25E-05	2.87E-04
Toluene	2.85E-07	6.38E-08	1.78E-05	2.85E-07	6.38E-08	1.78E-05
Vanadium	1.15E-02	4.41E-01	1.15E-01	1.18E-02	4.52E-01	1.18E-01

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-9

Exposure Point Concentrations in Spruce, Ludgate, and Menzies Lakes and Watershed Under Orimulsion High Load Conditions
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	For Evaluating Cancer Risk			For Evaluating Non-Cancer Effects		
	Water (mg/L)	Watershed Soil (0-1cm) (mg/kg)	Fish (mg/kg)	Water (mg/L)	Watershed Soil (0-1cm) (mg/kg)	Fish (mg/kg)
Mercury, elemental	NC	0.00E+00	NC	NC	0.00E+00	NC
Mercury, methyl	5.24E-10	1.18E-06	3.56E-03	8.27E-10	2.25E-06	5.62E-03
Mercury, divalent	2.13E-09	6.35E-05	NC	3.23E-09	1.26E-04	NC
1,4-Dichlorobenzene	1.02E-09	6.14E-09	2.90E-07	1.02E-09	6.14E-09	2.90E-07
Acenaphthene	4.05E-10	1.80E-07	2.46E-07	4.05E-10	1.80E-07	2.46E-07
Acenaphthylene	4.34E-10	2.20E-07	5.16E-06	4.83E-10	2.56E-07	5.75E-06
Anthracene	2.82E-11	9.44E-08	7.32E-08	2.82E-11	9.52E-08	7.34E-08
Arsenic	3.35E-07	6.85E-06	6.69E-06	3.39E-07	6.95E-06	6.77E-06
Benzene	1.08E-08	1.32E-09	2.67E-07	1.08E-08	1.32E-09	2.67E-07
Benzo(a)anthracene	4.74E-10	2.76E-06	2.42E-06	4.93E-10	3.01E-06	2.51E-06
Benzo(a)pyrene	1.13E-10	5.54E-07	1.12E-06	1.15E-10	5.95E-07	1.15E-06
Benzo(b)fluoranthene	1.97E-10	2.13E-06	1.96E-06	2.03E-10	2.31E-06	2.02E-06
Benzo(k)fluoranthene	6.21E-10	4.48E-06	6.18E-06	7.32E-10	5.91E-06	7.29E-06
Benzo(g,h,i)perylene	3.88E-09	1.96E-06	4.61E-05	4.32E-09	2.29E-06	5.14E-05
Beryllium	1.58E-08	6.97E-06	9.82E-07	2.14E-08	1.00E-05	1.33E-06
Cadmium	9.14E-09	4.75E-07	2.29E-06	9.42E-09	4.93E-07	2.36E-06
Chromium	1.30E-09	5.78E-04	2.48E-07	2.26E-09	1.16E-03	4.29E-07
Chromium VI	2.31E-07	3.11E-06	6.92E-07	2.33E-07	3.14E-06	6.98E-07
Chrysene	5.64E-10	2.20E-06	3.40E-06	6.03E-10	2.50E-06	3.63E-06
Dibenzo(a,h)anthracene	2.50E-10	2.35E-06	3.19E-06	2.63E-10	2.67E-06	3.37E-06
Fluoranthene	3.92E-10	1.78E-06	6.15E-06	4.00E-10	1.86E-06	6.29E-06
Fluorene	5.64E-09	2.69E-07	6.77E-06	5.66E-09	2.71E-07	6.79E-06
Formaldehyde	2.20E-08	7.32E-10	2.35E-08	2.20E-08	7.32E-10	2.35E-08
Indeno(1,2,3-cd)pyrene	1.31E-10	2.43E-06	1.72E-06	1.36E-10	2.69E-06	1.78E-06
Lead	2.26E-08	1.10E-05	1.81E-07	3.13E-08	1.64E-05	2.50E-07
Napthalene	5.45E-09	2.65E-07	1.17E-06	5.45E-09	2.65E-07	1.17E-06
Nickel	3.01E-04	1.36E-02	2.35E-02	3.09E-04	1.41E-02	2.41E-02
Phenanthrene	1.52E-10	1.26E-09	5.01E-07	1.52E-10	1.26E-09	5.01E-07
Pyrene	7.29E-09	3.69E-06	8.67E-05	8.13E-09	4.30E-06	9.67E-05
Toluene	1.47E-07	4.07E-08	9.19E-06	1.47E-07	4.07E-08	9.19E-06
Vanadium	1.19E-03	4.58E-02	1.19E-02	1.21E-03	4.70E-02	1.21E-02

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-10

Exposure Point Concentrations in Spruce, Ludgate, and Menzies Lakes and Watershed Under Natural Gas High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	For Evaluating Cancer Risk			For Evaluating Non-Cancer Effects		
	Water (mg/L)	Watershed Soil (0-1cm) (mg/kg)	Fish (mg/kg)	Water (mg/L)	Watershed Soil (0-1cm) (mg/kg)	Fish (mg/kg)
Mercury, elemental	NC	0.00E+00	NC	NC	0.00E+00	NC
Mercury, methyl	8.49E-12	1.82E-08	5.77E-05	1.32E-11	3.47E-08	8.95E-05
Mercury, divalent	3.49E-11	9.78E-07	NC	5.19E-11	1.95E-06	NC
1,4-Dichlorobenzene	8.92E-09	4.33E-08	2.53E-06	8.92E-09	4.33E-08	2.53E-06
Acenaphthene	1.86E-11	6.93E-09	1.13E-08	1.86E-11	6.94E-09	1.13E-08
Acenaphthylene	1.58E-09	7.85E-07	1.89E-05	1.76E-09	9.15E-07	2.10E-05
Anthracene	2.90E-11	8.38E-08	7.55E-08	2.91E-11	8.45E-08	7.56E-08
Arsenic	NA	0.00E+00	NA	NA	0.00E+00	NA
Benzene	1.40E-08	1.37E-09	3.46E-07	1.40E-08	1.37E-09	3.46E-07
Benzo(a)anthracene	1.07E-10	5.68E-07	5.44E-07	1.10E-10	6.19E-07	5.64E-07
Benzo(a)pyrene	6.01E-11	2.79E-07	5.98E-07	6.13E-11	3.00E-07	6.10E-07
Benzo(b)fluoranthene	5.51E-11	5.35E-07	5.48E-07	5.66E-11	5.80E-07	5.63E-07
Benzo(k)fluoranthene	1.76E-10	1.24E-06	1.76E-06	2.07E-10	1.63E-06	2.06E-06
Benzo(g,h,i)perylene	1.06E-09	5.25E-07	1.26E-05	1.18E-09	6.12E-07	1.40E-05
Beryllium	NA	0.00E+00	NA	NA	0.00E+00	NA
Cadmium	NA	0.00E+00	NA	NA	0.00E+00	NA
Chromium	NA	0.00E+00	NA	NA	0.00E+00	NA
Chromium VI	NA	0.00E+00	NA	NA	0.00E+00	NA
Chrysene	2.12E-10	7.74E-07	1.28E-06	2.26E-10	8.79E-07	1.36E-06
Dibenzo(a,h)anthracene	9.14E-11	8.33E-07	1.17E-06	9.61E-11	9.48E-07	1.23E-06
Fluoranthene	1.20E-10	4.98E-07	1.88E-06	1.22E-10	5.19E-07	1.92E-06
Fluorene	1.74E-09	7.67E-08	2.09E-06	1.74E-09	7.72E-08	2.09E-06
Formaldehyde	3.56E-07	9.61E-09	3.81E-07	3.56E-07	9.61E-09	3.81E-07
Indeno(1,2,3-cd)pyrene	5.63E-11	1.01E-06	7.38E-07	5.82E-11	1.11E-06	7.63E-07
Lead	NA	0.00E+00	NA	NA	0.00E+00	NA
Napthalene	4.77E-09	1.90E-07	1.03E-06	4.77E-09	1.90E-07	1.03E-06
Nickel	NA	0.00E+00	NA	NA	0.00E+00	No Q
Phenanthrene	1.38E-10	9.26E-10	4.55E-07	1.38E-10	9.26E-10	4.55E-07
Pyrene	4.40E-09	2.18E-06	5.24E-05	4.90E-09	2.54E-06	5.83E-05
Toluene	2.42E-08	5.42E-09	1.52E-06	2.42E-08	5.42E-09	1.52E-06
Vanadium	NA	0.00E+00	NA	NA	0.00E+00	NA

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 4-11
Pathway-Specific Parameters Used to Calculate Exposure Point Concentrations
Human Health Risk Assessment
Coleston Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Parameter	Value	Comment
BD (g/cm ³)	1.5	
q _{sw} (mL/cm ²)	0.2	
kp (yr ⁻¹)	18	
R (atm-m/3.mol-K)	8.21E-05	
mar (g/cm-s)	1.81E-04	
rair	1.20E-03	
rs	1.20E+03	
td (yr)	2.70	
Ta (K)	30	
T ₁ (yr)	298	
T ₂ (yr)	0	
Zs (cm)	30	
V _{dv} (cm/s)	1	untilled, for soil ingestion, for forage and soil which is consumed by livestock
k _{se} (yr ⁻¹)	20	tilled, for root veg. and aboveground produce consumed by humans and silage and grain consumed by livestock
P (cm/yr)	3	
I (cm/yr)	0	
RO (cm/yr)	144.0	(a)
E _a (cm/yr)	102.2	(a)
Rp (unitless)	52.8	(a)
	0.39	aboveground produce
	0.5	forage
	0.46	silage
	0.164	produce
	0.12	forage
	0.16	silage
	2.24	aboveground produce
	0.24	forage
	0.8	silage
	1	forage
	0.5	silage
	3	organics
	1	inorganics
	1.026	
	298	
	0.125	
	0.03	
	1	
	0.6	
	0.0011	
	5	(a)
	0.0012	
	1	
	0.4	
	4	

Table 4-11
 Pathway-Specific Parameters Used to Calculate Exposure Point Concentrations
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Parameter	Value	Comment
m_w (g/cm-s)	0.0169	
m_a (g/cm-s)	0.000181	
K_d (m/yr)	36500	
f_{lipid} (unitless)	0.07	
OC_{sed} (unitless)	0.04	
Type	lake	
A_w	5600279.1	Spruce Lake, Ludgate Lake and Menzies Lake combined (a)
A_l	204000	Assumed to be 1% of total Watershed
A_L	204000000	Watershed of Spruce Lake (a)
R_F	100	
K	0.39	
L_S	1.5	
C	0.1	
P_F	1	
V_{fx}	1.83E+07	Inflow into Spruce Lake Reservoir (a)
dwc	20	Max of Spruce Lake (a)
TSS	10	
u	1.35636	
F_w (unitless)	1.4	
V_{G_2} (unitless)	0.2	anions
V_{G_3} (unitless)	0.6	cations and organics
MF (unitless)	0.01	log Kow>4
B_s (unitless)	1	log Kow<4
Forage Ingestion Rate (kg/day)	0.01	log Kow>4
Grain Ingestion Rate (kg/day)	1	log Kow<4
Silage Ingestion Rate (kg/day)	1	
Soil Ingestion Rate (kg/day)	1	
	8.8	Beef cattle
	13.2	Dairy cattle
	0.47	Beef cattle
	3	Dairy cattle
	3.3	Pig
	0.2	Poultry
	2.5	Beef cattle
	4.1	Dairy cattle
	1.4	Pig
	0.5	Beef cattle
	0.4	Dairy cattle
	0.37	Pig
	0.022	Poultry

Source
 U.S EPA, 1998a, unless otherwise noted.

Table 4-12

Exposure Point Concentrations in Produce Under Fuel Oil High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Produce - above ground (mg/kg)				Produce - root (mg/kg)			
	For Evaluating Cancer Risk		For Evaluating Non-Cancer Effects		For Evaluating Cancer Risk		For Evaluating Non-Cancer Effects	
	Maximum Hypothetical Residence	Saint John Northeast Farm	Maximum Hypothetical Residence	Saint John Northeast Farm	Maximum Hypothetical Residence	Saint John Northeast Farm	Maximum Hypothetical Residence	Saint John Northeast Farm
Mercury, elemental	NC	NC	NC	NC	NC	NC	NC	NC
Mercury, methyl	1.57E-06	3.79E-08	1.79E-06	2.62E-08	7.65E-07	2.42E-08	1.53E-06	4.82E-08
Mercury, divalent	1.03E-05	3.87E-07	1.58E-05	3.15E-07	1.37E-05	4.33E-07	2.74E-05	8.65E-07
1,4-Dichlorobenzene	2.15E-06	7.17E-08	2.16E-06	4.10E-08	8.86E-05	2.95E-06	8.88E-05	2.96E-06
Acenaphthene	1.36E-06	4.55E-08	1.37E-06	2.62E-08	3.75E-05	1.25E-06	3.80E-05	1.27E-06
Acenaphthylene	3.30E-07	9.11E-09	3.47E-07	5.43E-09	2.81E-08	6.68E-10	3.62E-08	8.62E-10
Anthracene	1.76E-07	5.84E-09	1.86E-07	3.54E-09	4.79E-08	1.59E-09	5.08E-08	1.69E-09
Arsenic	5.54E-04	1.03E-05	5.58E-04	5.54E-06	1.62E-05	2.60E-07	2.18E-05	3.48E-07
Benzo(a)anthracene	8.35E-07	2.78E-08	8.35E-07	1.59E-08	9.90E-06	3.30E-07	9.90E-06	3.30E-07
Benzo(a)pyrene	8.14E-07	2.20E-08	8.31E-07	1.29E-08	1.77E-07	5.67E-09	1.95E-07	6.22E-09
Benzo(b)fluoranthene	1.11E-06	2.73E-08	1.11E-06	1.58E-08	2.24E-08	5.51E-10	2.41E-08	5.92E-10
Benzo(k)fluoranthene	7.91E-07	2.13E-08	7.96E-07	1.23E-08	9.63E-08	3.01E-09	1.05E-07	3.27E-09
Benzo(g,h,i)perylene	3.66E-06	9.10E-08	3.71E-06	5.34E-08	2.56E-07	5.94E-09	3.43E-07	7.94E-09
Beryllium	5.13E-07	3.53E-08	6.61E-07	2.02E-08	2.50E-07	5.95E-09	3.23E-07	7.68E-09
Cadmium	2.01E-05	3.73E-07	2.05E-05	2.04E-07	2.57E-07	4.11E-09	5.03E-07	8.05E-09
Chromium VI	2.59E-05	4.53E-07	3.31E-05	3.09E-07	5.72E-06	9.15E-08	9.38E-06	1.50E-07
Chrysene	9.24E-04	1.71E-05	9.64E-04	9.51E-06	3.62E-05	5.79E-07	7.24E-05	1.16E-06
Dibenz(a,h)anthracene	2.60E-04	4.83E-06	2.61E-04	2.59E-06	3.20E-06	5.12E-08	3.91E-06	6.26E-08
Fluorene	8.95E-07	2.46E-08	9.14E-07	1.44E-08	1.42E-07	4.36E-09	1.64E-07	5.01E-09
Formaldehyde	5.73E-06	1.31E-07	5.74E-06	6.92E-08	1.60E-11	2.58E-13	1.83E-11	2.94E-13
Indeno(1,2,3-cd)pyrene	3.53E-07	1.14E-08	3.72E-07	6.52E-09	2.64E-07	8.76E-09	2.80E-07	9.30E-09
Lead	5.26E-07	1.53E-08	5.27E-07	8.74E-09	4.44E-08	1.45E-09	4.48E-08	1.46E-09
Naphthalene	2.74E-04	9.12E-06	2.74E-04	5.20E-06	2.86E-03	9.52E-05	2.86E-03	9.52E-05
Nickel	7.33E-06	2.53E-07	7.33E-06	1.30E-07	1.37E-07	2.20E-09	1.52E-07	2.43E-09
Phenanthrene	9.76E-03	1.79E-04	1.08E-02	1.05E-04	6.87E-04	1.10E-05	1.35E-03	2.16E-05
Pyrene	6.34E-05	2.11E-06	6.37E-05	1.21E-06	1.20E-03	3.99E-05	1.20E-03	4.01E-05
Toluene	2.69E-01	4.98E-03	2.78E-01	5.11E-03	1.17E-02	1.88E-04	1.88E-02	3.00E-04
Vanadium	1.05E-07	3.49E-09	1.05E-07	2.00E-09	1.72E-08	5.72E-10	1.73E-08	5.72E-10
	5.55E-06	1.53E-07	5.83E-06	9.13E-08	4.72E-07	1.12E-08	6.09E-07	1.45E-08
	2.78E-05	9.27E-07	2.78E-05	5.29E-07	5.84E-04	1.94E-05	5.84E-04	1.94E-05
	1.73E+00	3.21E-02	1.75E+00	1.74E-02	2.58E-02	4.12E-04	3.91E-02	6.26E-04

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-12
Exposure Point Concentrations in Produce Under Fuel Oil High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Produce - forage (mg/kg)			Produce - silage (mg/kg)			Produce - grain (mg/kg)		
	For Evaluating Cancer Risk	For Evaluating Non-Cancer Effects	Northeast Farm	For Evaluating Cancer Risk	For Evaluating Non-Cancer Effects	Northeast Farm	For Evaluating Cancer Risk	For Evaluating Non-Cancer Effects	Northeast Farm
Mercury, elemental	0.00E+00	0.00E+00	Northeast Farm	0.00E+00	0.00E+00	Northeast Farm	0.00E+00	0.00E+00	Northeast Farm
Mercury, methyl	2.01E-07	2.01E-07		5.92E-08	5.92E-08		0.00E+00	0.00E+00	
Mercury, divalent	7.63E-07	7.63E-07		2.35E-07	2.35E-07		0.00E+00	0.00E+00	
1,4-Dichlorobenzene	2.46E-09	2.46E-09		4.09E-08	4.10E-08		4.09E-08	4.10E-08	
Acenaphthene	3.72E-08	3.72E-08		2.58E-08	2.62E-08		2.58E-08	2.61E-08	
Acenaphthylene	1.25E-07	1.27E-07		4.95E-08	4.98E-08		8.03E-10	1.04E-09	
Anthracene	1.00E-08	1.01E-08		3.37E-09	3.58E-09		3.33E-09	3.54E-09	
Arsenic	6.11E-05	6.11E-05		1.84E-05	1.86E-05		6.44E-07	8.63E-07	
Benzene	8.06E-10	8.06E-10		1.59E-08	1.59E-08		1.59E-08	1.59E-08	
Benzofluoranthracene	2.41E-07	2.46E-07		7.48E-08	7.51E-08		3.11E-09	3.41E-09	
Benzofluoranthene	2.62E-07	2.62E-07		9.49E-08	9.49E-08		2.85E-10	3.06E-10	
Benzofluoranthene	2.61E-07	2.63E-07		9.87E-08	9.88E-08		1.05E-09	1.14E-09	
Benzofluoranthene	9.16E-07	9.29E-07		3.32E-07	3.33E-07		2.14E-09	2.85E-09	
Benzofluoranthene	7.99E-07	8.14E-07		3.49E-07	3.51E-07		7.16E-09	9.24E-09	
Beryllium	2.35E-06	2.42E-06		6.60E-07	6.74E-07		1.51E-08	2.96E-08	
Cadmium	2.25E-06	2.28E-06		7.71E-07	9.54E-07		2.87E-07	4.70E-07	
Chromium	1.09E-04	1.20E-04		2.95E-05	3.01E-05		5.32E-07	1.06E-06	
Chromium VI	2.86E-05	2.86E-05		8.46E-06	8.47E-06		4.70E-08	5.75E-08	
Chrysene	3.11E-07	3.17E-07		1.13E-07	1.13E-07		2.28E-09	2.62E-09	
Dibenzofluoranthracene	1.85E-06	1.85E-06		7.89E-07	7.89E-07		6.32E-10	7.21E-10	
Fluoranthene	1.01E-07	1.04E-07		1.33E-08	1.37E-08		5.72E-09	6.07E-09	
Fluorene	1.98E-07	1.98E-07		6.82E-08	6.82E-08		2.52E-09	2.54E-09	
Formaldehyde	2.65E-07	2.65E-07		5.20E-06	5.20E-06		5.20E-06	5.20E-06	
Indeno(1,2,3-cd)pyrene	6.57E-06	6.57E-06		3.11E-06	3.11E-06		3.98E-10	4.40E-10	
Lead	1.31E-03	1.47E-03		3.16E-04	3.45E-04		3.03E-05	5.94E-05	
Naphthalene	3.53E-07	3.53E-07		1.20E-06	1.21E-06		1.20E-06	1.21E-06	
Nickel	2.93E-02	2.93E-02		8.80E-03	9.04E-03		4.14E-04	6.62E-04	
Phenanthrene	1.19E-10	1.19E-10		1.99E-09	2.00E-09		1.99E-09	2.00E-09	
Pyrene	2.10E-06	2.13E-06		8.33E-07	8.36E-07		1.35E-08	1.74E-08	
Toluene	2.81E-08	2.81E-08		5.29E-07	5.29E-07		5.29E-07	5.29E-07	
Vanadium	1.88E-01	1.88E-01		5.55E-02	5.57E-02		4.17E-04	6.32E-04	

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-13
Exposure Point Concentrations in Produce Under Orlimulsion High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Produce - above ground (mg/kg)						Produce - root (mg/kg)					
	For Evaluating Cancer Risk			For Evaluating Non-Cancer Effects			For Evaluating Cancer Risk			For Evaluating Non-Cancer Effects		
	Maximum Hypothetical Residence	Saint John	Northeast Farm	Maximum Hypothetical Residence	Saint John	Northeast Farm	Maximum Hypothetical Residence	Saint John	Northeast Farm	Maximum Hypothetical Residence	Saint John	Northeast Farm
Mercury, elemental	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC
Mercury, methyl	2.94E-07	7.39E-09	4.01E-09	3.36E-07	9.07E-09	4.91E-09	1.43E-07	5.67E-09	3.05E-09	2.85E-07	1.13E-08	6.09E-09
Mercury, divalent	1.92E-06	1.02E-07	5.29E-08	2.95E-06	1.43E-07	7.49E-08	2.56E-06	1.01E-07	5.46E-08	5.11E-06	2.03E-07	1.09E-07
1,4-Dichlorobenzene	9.01E-07	3.87E-08	2.08E-08	9.03E-07	3.89E-08	2.09E-08	3.71E-05	1.59E-06	8.56E-07	3.72E-05	1.60E-06	8.58E-07
Acenaphthene	5.27E-07	2.29E-08	1.23E-08	5.34E-07	2.32E-08	1.24E-08	1.46E-05	6.27E-07	3.37E-07	1.48E-05	6.35E-07	3.41E-07
Acenaphthylene	1.29E-07	4.20E-09	2.22E-09	1.36E-07	4.36E-09	2.30E-09	1.10E-08	2.67E-10	1.48E-10	1.42E-08	3.45E-10	1.92E-10
Anthracene	6.83E-08	2.94E-09	1.58E-09	7.26E-08	3.12E-09	1.67E-09	1.86E-08	8.01E-10	4.30E-10	1.98E-08	8.51E-10	4.57E-10
Arsenic	5.13E-05	1.13E-06	5.50E-07	5.17E-05	1.14E-06	5.53E-07	1.50E-06	2.56E-08	1.30E-08	2.02E-06	3.43E-08	1.74E-08
Benzene	1.24E-06	5.33E-08	2.86E-08	1.24E-06	5.33E-08	2.86E-08	1.47E-05	6.33E-07	3.40E-07	1.47E-05	6.33E-07	3.40E-07
Benzo(a)anthracene	3.18E-07	9.82E-09	5.24E-09	3.24E-07	1.01E-08	5.38E-09	6.90E-08	2.78E-09	1.50E-09	7.57E-08	3.05E-09	1.64E-09
Benzo(a)pyrene	4.67E-07	1.23E-08	6.59E-09	4.67E-07	1.23E-08	6.59E-09	9.41E-09	2.45E-10	1.35E-10	1.01E-08	2.63E-10	1.45E-10
Benzo(b)fluoranthene	3.33E-07	1.03E-08	5.46E-09	3.35E-07	1.04E-08	5.51E-09	4.04E-08	1.58E-09	8.50E-10	4.39E-08	1.71E-09	9.24E-10
Benzo(k)fluoranthene	1.54E-06	4.13E-08	2.21E-08	1.56E-06	4.19E-08	2.24E-08	1.08E-07	2.50E-09	1.40E-09	1.44E-07	3.35E-09	1.87E-09
Benzo(g,h,i)perylene	2.01E-07	2.34E-08	1.11E-08	2.59E-07	2.49E-08	1.19E-08	9.81E-08	2.39E-09	1.33E-09	1.27E-07	3.09E-09	1.71E-09
Beryllium	2.95E-06	6.49E-08	3.16E-08	3.01E-06	6.60E-08	3.21E-08	3.77E-08	6.41E-10	3.26E-10	7.38E-08	1.26E-09	6.38E-10
Cadmium	2.42E-06	4.83E-08	2.39E-08	3.09E-06	5.97E-08	2.96E-08	5.35E-07	9.10E-09	4.63E-09	8.77E-07	1.49E-08	7.58E-09
Chromium	1.24E-04	2.72E-06	1.32E-06	1.29E-04	2.81E-06	1.37E-06	4.86E-06	8.28E-08	4.21E-08	9.73E-06	1.66E-07	8.41E-08
Chromium VI	3.50E-05	7.72E-07	3.75E-07	3.51E-05	7.74E-07	3.76E-07	4.30E-07	7.32E-09	3.72E-09	5.26E-07	8.95E-09	4.55E-09
Chrysene	3.49E-07	1.12E-08	5.96E-09	3.57E-07	1.15E-08	6.11E-09	5.54E-08	2.09E-09	1.13E-09	6.37E-08	2.40E-09	1.30E-09
Dibenz(a,h)anthracene	2.26E-06	7.01E-08	3.40E-08	2.27E-06	7.01E-08	3.41E-08	6.33E-12	1.08E-13	5.51E-14	7.22E-12	1.24E-13	6.29E-14
Fluoranthene	1.38E-07	5.68E-09	3.04E-09	1.45E-07	5.99E-09	3.21E-09	1.03E-07	4.41E-09	2.37E-09	1.09E-07	4.68E-09	2.51E-09
Fluorene	2.05E-07	7.27E-09	3.84E-09	2.06E-07	7.28E-09	3.85E-09	1.73E-08	7.18E-10	3.86E-10	1.74E-08	7.23E-10	3.89E-10
Formaldehyde	7.51E-06	3.23E-07	1.73E-07	7.51E-06	3.23E-07	1.73E-07	7.84E-05	3.37E-06	1.81E-06	7.84E-05	3.37E-06	1.81E-06
Indeno(1,2,3-cd)pyrene	2.89E-06	1.60E-07	7.77E-08	2.90E-06	1.60E-07	7.77E-08	5.41E-08	9.25E-10	4.71E-10	5.99E-08	1.02E-09	5.21E-10
Lead	4.77E-06	1.03E-07	5.02E-08	5.26E-06	1.11E-07	5.45E-08	3.36E-07	5.71E-09	2.91E-09	6.59E-07	1.12E-08	5.70E-09
Naphthalene	8.40E-06	3.61E-07	1.94E-07	8.44E-06	3.63E-07	1.95E-07	1.59E-04	6.83E-06	3.67E-06	1.60E-04	6.87E-06	3.69E-06
Nickel	4.78E-02	1.05E-03	5.09E-04	4.93E-02	1.07E-03	5.22E-04	2.08E-03	3.55E-05	1.80E-05	3.33E-03	5.67E-05	2.88E-05
Phenanthrene	4.08E-08	1.75E-09	9.41E-10	4.10E-08	1.76E-09	9.45E-10	6.69E-09	2.88E-10	1.54E-10	6.72E-09	2.89E-10	1.55E-10
Pyrene	2.17E-06	7.05E-08	3.72E-08	2.28E-06	7.32E-08	3.87E-08	1.84E-07	4.49E-09	2.49E-09	2.38E-07	5.80E-09	3.22E-09
Toluene	1.81E-05	7.80E-07	4.18E-07	1.81E-05	7.80E-07	4.19E-07	3.80E-04	1.64E-05	8.78E-06	3.81E-04	1.64E-05	8.78E-06
Vanadium	2.03E-01	4.46E-03	2.17E-03	2.05E-01	4.50E-03	2.19E-03	3.02E-03	5.14E-05	2.61E-05	4.58E-03	7.80E-05	3.97E-05

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-13
Exposure Point Concentrations in Produce Under Oromulsion High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Produce - forage (mg/kg)		Produce - silage (mg/kg)		Produce - grain (mg/kg)	
	For Evaluating Cancer Risk	For Evaluating Non-Cancer Effects	For Evaluating Cancer Risk	For Evaluating Non-Cancer Effects	For Evaluating Cancer Risk	For Evaluating Non-Cancer Effects
	Northeast Farm	Northeast Farm	Northeast Farm	Northeast Farm	Northeast Farm	Northeast Farm
Mercury, elemental	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Mercury, methyl	3.46E-08	3.46E-08	1.02E-08	1.02E-08	0.00E+00	0.00E+00
Mercury, divalent	1.43E-07	1.43E-07	4.61E-08	4.61E-08	0.00E+00	0.00E+00
1,4-Dichlorobenzene	1.25E-09	1.25E-09	2.08E-08	2.08E-08	2.08E-08	2.08E-08
Aceaphthylene	1.76E-08	1.76E-08	1.24E-08	1.24E-08	1.23E-08	1.23E-08
Acenaphthylene	7.82E-08	7.82E-08	3.43E-08	3.43E-08	3.03E-10	3.91E-10
Anthracene	4.74E-09	4.78E-09	1.60E-09	1.70E-09	1.57E-09	1.67E-09
Arsenic	6.10E-06	6.11E-06	1.83E-06	1.85E-06	5.86E-08	7.85E-08
Benzene	1.45E-09	1.45E-09	2.86E-08	2.86E-08	2.86E-08	2.86E-08
Benzo(a)anthracene	1.31E-07	1.34E-07	4.66E-08	4.67E-08	1.43E-09	1.57E-09
Benzo(a)pyrene	1.47E-07	1.47E-07	5.93E-08	5.93E-08	1.19E-10	1.28E-10
Benzo(b)fluoranthene	1.66E-07	1.67E-07	7.00E-08	7.00E-08	5.16E-10	5.61E-10
Benzo(k)fluoranthene	5.25E-07	5.30E-07	2.13E-07	2.14E-07	8.49E-10	1.13E-09
Benzo(g,h,i)perylene	6.02E-07	6.08E-07	2.78E-07	2.79E-07	2.71E-09	3.50E-09
Beryllium	3.69E-07	3.79E-07	1.04E-07	1.06E-07	2.17E-09	4.25E-09
Cadmium	2.22E-07	2.24E-07	7.51E-08	9.19E-08	2.63E-08	4.31E-08
Chromium	1.57E-05	1.71E-05	4.28E-06	4.35E-06	7.01E-08	1.40E-07
Chromium VI	4.16E-06	4.16E-06	1.23E-06	1.23E-06	6.20E-09	7.59E-09
Chrysene	1.85E-07	1.87E-07	7.54E-08	7.55E-08	1.03E-09	1.18E-09
Dibenzo(a,h)anthracene	1.25E-06	1.25E-06	5.68E-07	5.68E-07	2.45E-10	2.80E-10
Fluoranthene	5.04E-08	5.20E-08	7.84E-09	8.01E-09	2.71E-09	2.87E-09
Fluorene	1.19E-07	1.19E-07	4.65E-08	4.65E-08	1.17E-09	1.18E-09
Formaldehyde	8.84E-09	8.84E-09	1.73E-07	1.73E-07	1.73E-07	1.73E-07
Indeno(1,2,3-cd)pyrene	5.01E-06	5.01E-06	2.43E-06	2.43E-06	1.54E-10	1.71E-10
Lead	6.73E-07	7.50E-07	1.65E-07	1.79E-07	1.45E-08	2.85E-08
Naphthalene	5.69E-08	5.69E-08	1.94E-07	1.95E-07	1.94E-07	1.95E-07
Nickel	5.59E-03	5.60E-03	1.68E-03	1.72E-03	7.21E-05	1.15E-04
Phenanthrene	5.66E-11	5.66E-11	9.42E-10	9.46E-10	9.41E-10	9.45E-10
Pyrene	1.31E-06	1.33E-06	5.77E-07	5.79E-07	5.09E-09	6.57E-09
Toluene	2.23E-08	2.23E-08	4.18E-07	4.19E-07	4.18E-07	4.18E-07
Vanadium	2.38E-02	2.38E-02	7.02E-03	7.04E-03	4.79E-05	7.27E-05

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 4-14
Exposure Point Concentrations in Produce Under Natural Gas High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Produce - above ground (mg/kg)					Produce - root (mg/kg)				
	For Evaluating Cancer Risk			For Evaluating Non-Cancer Effects		For Evaluating Cancer Risk			For Evaluating Non-Cancer Effects	
	Maximum Hypothetical Residence	Saint John	Northeast Farm	Maximum Hypothetical Residence	Northeast Farm	Maximum Hypothetical Residence	Saint John	Northeast Farm	Maximum Hypothetical Residence	Northeast Farm
Mercury, elemental	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC
Mercury, methyl	4.35E-09	1.05E-10	6.13E-11	4.97E-09	1.25E-10	7.27E-11	6.71E-11	3.84E-11	4.24E-09	1.34E-10
Mercury, divalent	2.85E-08	1.07E-09	5.96E-10	4.38E-08	1.56E-09	8.73E-10	1.20E-09	6.88E-10	7.60E-08	2.40E-09
1,4-Dichlorobenzene	6.23E-06	2.08E-07	1.18E-07	6.25E-06	2.08E-07	1.19E-07	8.54E-06	4.88E-06	2.57E-04	8.57E-06
Acenaphthene	1.99E-08	6.68E-10	3.81E-10	2.02E-08	6.76E-10	3.86E-10	1.84E-08	1.05E-08	5.58E-07	1.86E-08
Acenaphthylene	4.05E-07	1.12E-08	6.38E-09	4.28E-07	1.17E-08	6.67E-09	8.19E-10	4.82E-10	4.45E-08	1.06E-09
Anthracene	5.95E-08	1.98E-09	1.13E-09	6.31E-08	2.10E-09	1.20E-09	5.40E-10	3.08E-10	1.72E-08	5.73E-10
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	1.27E-06	4.23E-08	2.41E-08	1.27E-06	4.23E-08	2.41E-08	5.02E-07	2.86E-07	1.51E-05	5.02E-07
Benzo(a)anthracene	6.29E-08	1.70E-09	9.75E-10	6.42E-08	1.74E-09	9.99E-10	4.38E-10	2.51E-10	1.50E-08	4.81E-10
Benzo(a)pyrene	2.08E-07	5.14E-09	2.98E-09	2.08E-07	5.15E-09	2.98E-09	1.04E-10	6.08E-11	4.53E-09	1.11E-10
Benzo(b)fluoranthene	8.01E-08	2.15E-09	1.23E-09	8.06E-08	2.17E-09	1.24E-09	9.75E-09	1.75E-10	1.06E-08	3.32E-10
Benzo(k)fluoranthene	3.71E-07	9.22E-09	5.33E-09	3.76E-07	9.34E-09	5.41E-09	6.02E-10	3.55E-10	3.47E-08	8.04E-10
Benzo(g,h,i)perylene	4.71E-08	3.25E-09	1.66E-09	6.08E-08	3.57E-09	1.85E-09	5.47E-10	3.22E-10	2.97E-08	7.06E-10
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	1.16E-07	3.20E-09	1.83E-09	1.19E-07	3.28E-09	1.88E-09	5.67E-10	3.26E-10	2.13E-08	6.52E-10
Dibenz(a,h)anthracene	7.12E-07	1.63E-08	8.58E-09	7.13E-07	1.63E-08	8.59E-09	3.20E-14	1.77E-14	2.27E-12	3.65E-14
Fluoranthene	3.77E-08	1.22E-09	6.96E-10	3.97E-08	1.28E-09	7.33E-10	9.35E-10	5.34E-10	2.99E-08	9.92E-10
Fluorene	5.68E-08	1.65E-09	9.42E-10	5.69E-08	1.66E-09	9.45E-10	1.55E-10	8.92E-11	4.83E-09	1.57E-10
Formaldehyde	9.65E-05	3.22E-06	1.83E-06	9.65E-05	3.22E-06	1.83E-06	3.36E-05	1.92E-05	1.01E-03	3.36E-05
Indeno(1,2,3-cd)pyrene	1.06E-06	3.67E-08	1.89E-08	1.06E-06	3.67E-08	1.89E-08	3.19E-10	1.76E-10	2.20E-08	3.53E-10
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	5.89E-06	1.96E-07	1.12E-07	5.92E-06	1.97E-07	1.13E-07	3.71E-06	2.12E-06	1.12E-04	3.73E-06
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	2.93E-08	9.75E-10	5.56E-10	2.94E-08	9.79E-10	5.59E-10	1.60E-10	9.13E-11	4.80E-09	1.61E-10
Pyrene	1.13E-08	3.10E-08	1.77E-08	1.18E-08	3.24E-08	1.85E-08	2.27E-09	1.34E-09	1.24E-07	2.94E-09
Toluene	2.37E-06	7.88E-08	4.50E-08	2.37E-06	7.88E-08	4.50E-08	1.65E-06	9.44E-07	4.97E-05	1.65E-06
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 4-14
Exposure Point Concentrations in Produce Under Natural Gas High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Produce - forage (mg/kg)			Produce - silage (mg/kg)		Produce - grain (mg/kg)	
	For Evaluating Cancer Risk	For Evaluating Non-Cancer Effects	Northeast Farm	For Evaluating Cancer Risk	For Evaluating Non-Cancer Effects	For Evaluating Cancer Risk	For Evaluating Non-Cancer Effects
Mercury, elemental	0.00E+00	0.00E+00	Northeast Farm	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Mercury, methyl	5.57E-10	5.57E-10		1.64E-10	1.64E-10	0.00E+00	0.00E+00
Mercury, divalent	2.12E-09	2.12E-09		6.53E-10	6.53E-10	0.00E+00	0.00E+00
1,4-Dichlorobenzene	7.11E-09	7.11E-09		1.18E-07	1.18E-07	1.18E-07	1.18E-07
Acenaphthene	5.46E-10	5.47E-10		3.80E-10	3.85E-10	3.79E-10	3.83E-10
Acenaphthylene	1.53E-07	1.56E-07		6.08E-08	6.11E-08	9.85E-10	1.27E-09
Anthracene	3.39E-09	3.42E-09		1.14E-09	1.21E-09	1.13E-09	1.20E-09
Arsenic	0.00E+00	0.00E+00		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Benzene	1.23E-09	1.23E-09		2.41E-08	2.41E-08	2.41E-08	2.41E-08
Benzo(a)anthracene	1.86E-08	1.90E-08		5.78E-09	5.80E-09	2.40E-10	2.64E-10
Benzo(a)pyrene	4.93E-08	4.94E-08		1.79E-08	1.79E-08	5.36E-11	5.76E-11
Benzo(b)fluoranthene	2.64E-08	2.66E-08		9.99E-09	1.00E-08	1.06E-10	1.15E-10
Benzo(k)fluoranthene	9.28E-08	9.41E-08		3.36E-08	3.37E-08	2.16E-10	2.89E-10
Benzo(g,h,i)perylene	7.34E-08	7.48E-08		3.21E-08	3.22E-08	6.58E-10	8.49E-10
Beryllium	0.00E+00	0.00E+00		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium	0.00E+00	0.00E+00		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chromium	0.00E+00	0.00E+00		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chromium VI	0.00E+00	0.00E+00		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chrysene	4.04E-08	4.12E-08		1.47E-08	1.47E-08	2.96E-10	3.41E-10
Dibenzo(a,h)anthracene	2.30E-07	2.30E-07		9.80E-08	9.80E-08	7.85E-11	8.96E-11
Fluoranthene	2.13E-08	1.11E-08		1.42E-09	1.46E-09	6.11E-10	6.48E-10
Fluorene	9.36E-08	9.36E-08		7.35E-09	7.36E-09	2.72E-10	2.74E-10
Formaldehyde	9.53E-07	9.53E-07		1.83E-06	1.83E-06	1.83E-06	1.83E-06
Indeno(1,2,3-cd)pyrene	0.00E+00	0.00E+00		4.52E-07	4.52E-07	5.77E-11	6.38E-11
Lead	0.00E+00	0.00E+00		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Naphthalene	3.28E-08	3.28E-08		1.12E-07	1.13E-07	1.12E-07	1.13E-07
Nickel	0.00E+00	0.00E+00		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Phenanthrene	3.34E-11	3.34E-11		5.56E-10	5.59E-10	5.56E-10	5.59E-10
Pyrene	4.26E-07	4.32E-07		1.69E-07	1.70E-07	2.73E-09	3.53E-09
Toluene	2.39E-09	2.39E-09		4.50E-08	4.50E-08	4.50E-08	4.50E-08
Vanadium	0.00E+00	0.00E+00		0.00E+00	0.00E+00	0.00E+00	0.00E+00

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 4-15
Exposure Point Concentrations in Animal Products Under Fuel Oil High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	For Evaluating Cancer Risk (mg/kg)					For Evaluating Non-Cancer Effects (mg/kg)				
	Beef	Milk	Chicken	Eggs	Pork	Beef	Milk	Chicken	Eggs	Pork
Mercury, elemental	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC
Mercury, methyl	2.49E-09	1.32E-09	2.00E-10	2.00E-10	5.19E-12	3.38E-09	1.63E-09	3.80E-10	3.80E-10	9.48E-12
Mercury, divalent	3.94E-07	1.48E-07	7.16E-08	7.16E-08	1.72E-09	7.45E-07	2.70E-07	1.42E-07	1.42E-07	3.41E-09
1,4-Dichlorobenzene	9.48E-12	6.67E-12	5.58E-13	2.24E-10	1.53E-11	9.50E-12	6.68E-12	5.60E-13	2.24E-10	1.53E-11
Acenaphthene	1.15E-10	5.48E-11	1.70E-12	6.78E-10	5.33E-11	1.15E-10	5.50E-11	1.71E-12	6.83E-10	5.38E-11
Acenaphthylene	3.36E-09	1.55E-09	9.71E-12	3.89E-09	4.63E-10	3.44E-09	1.58E-09	1.14E-11	4.55E-09	5.07E-10
Anthracene	1.09E-10	4.56E-11	1.65E-12	6.60E-10	4.67E-11	1.10E-10	4.63E-11	1.69E-12	6.74E-10	4.78E-11
Arsenic	1.19E-06	5.36E-08	NA	NA	NA	1.19E-06	5.37E-08	NA	NA	NA
Benzene	1.87E-13	1.35E-13	8.65E-15	3.47E-12	3.11E-13	1.87E-13	1.35E-13	8.65E-15	3.47E-12	3.11E-13
Benzo(a)anthracene	4.48E-08	1.76E-08	6.00E-10	2.40E-07	1.70E-08	4.69E-08	1.82E-08	6.54E-10	2.62E-07	1.84E-08
Benzo(a)pyrene	9.45E-08	4.33E-08	2.99E-10	1.20E-07	1.32E-08	9.53E-08	4.36E-08	3.21E-10	1.29E-07	1.37E-08
Benzo(b)fluoranthene	1.42E-07	5.92E-08	1.42E-09	5.70E-07	4.32E-08	1.46E-07	6.04E-08	1.54E-09	6.19E-07	4.63E-08
Benzo(k)fluoranthene	4.34E-07	1.90E-07	2.81E-09	1.13E-06	9.49E-08	4.65E-07	1.99E-07	3.70E-09	1.48E-06	1.18E-07
Benzo(g,h,i)perylene	2.23E-08	1.02E-08	8.66E-11	3.47E-08	3.73E-09	2.31E-08	1.05E-08	1.01E-10	4.06E-08	4.12E-09
Beryllium	3.01E-08	3.60E-11	NA	NA	NA	3.42E-08	3.94E-11	NA	NA	NA
Cadmium	2.73E-09	2.24E-10	9.99E-09	2.36E-10	5.05E-10	2.82E-09	2.34E-10	1.40E-08	3.31E-10	6.75E-10
Chromium	9.59E-06	3.20E-06	NA	NA	NA	1.40E-05	4.26E-06	NA	NA	NA
Chromium VI	1.52E-06	6.24E-07	NA	NA	NA	1.52E-06	6.24E-07	NA	NA	NA
Chrysene	5.72E-08	2.39E-08	5.47E-10	2.18E-07	1.67E-08	6.00E-08	2.47E-08	6.22E-10	2.48E-07	1.87E-08
Dibenzo(a,h)anthracene	1.70E-06	7.96E-07	3.04E-09	1.21E-06	1.96E-07	1.72E-06	7.99E-07	3.46E-09	1.38E-06	2.07E-07
Fluoranthene	5.63E-09	2.06E-09	1.01E-10	4.03E-08	2.66E-09	5.84E-09	2.14E-09	1.05E-10	4.20E-08	2.77E-09
Fluorene	7.58E-10	3.52E-10	1.94E-12	7.76E-10	9.27E-11	7.59E-10	3.52E-10	1.95E-12	7.81E-10	9.30E-11
Formaldehyde	9.84E-13	7.08E-13	4.54E-14	1.82E-11	1.64E-12	9.84E-13	7.08E-13	4.54E-14	1.82E-11	1.64E-12
Indeno(1,2,3-cd)pyrene	1.38E-05	6.55E-06	7.30E-09	2.92E-06	1.28E-06	1.38E-05	6.56E-06	8.07E-09	3.23E-06	1.30E-06
Lead	4.81E-06	5.40E-06	NA	NA	1.19E-06	5.80E-06	6.34E-06	NA	NA	1.71E-06
Naphthalene	4.20E-10	2.53E-10	1.21E-11	4.84E-09	4.27E-10	4.21E-10	2.54E-10	1.21E-11	4.86E-09	4.29E-10
Nickel	1.75E-03	4.34E-04	NA	NA	NA	1.76E-03	4.36E-04	NA	NA	NA
Phenanthrene	6.79E-12	4.58E-12	3.01E-13	1.20E-10	1.06E-11	6.82E-12	4.59E-12	3.02E-13	1.21E-10	1.07E-11
Pyrene	5.64E-08	2.60E-08	1.63E-10	6.54E-08	7.78E-09	5.78E-08	2.66E-08	1.91E-10	7.65E-08	8.51E-09
Toluene	2.14E-11	1.53E-11	9.80E-13	3.92E-10	3.52E-11	2.14E-11	1.53E-11	9.80E-13	3.92E-10	3.52E-11
Vanadium	4.64E-03	5.52E-03	NA	NA	NA	4.65E-03	5.53E-03	NA	NA	NA

Notes:
NC - Not calculated. This compound is not assumed to be present in this medium.
NA - No biotransfer factor is available for this compound for this medium.

Table 4-16
Exposure Point Concentrations in Animal Products Under Orimulsion High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	For Evaluating Cancer Risk (mg/kg)					For Evaluating Non-Cancer Effects (mg/kg)				
	Beef	Milk	Chicken	Eggs	Pork	Beef	Milk	Chicken	Eggs	Pork
Mercury, elemental	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC
Mercury, methyl	4.76E-10	2.44E-10	4.41E-11	4.41E-11	1.12E-12	6.73E-10	3.13E-10	8.38E-11	8.38E-11	2.07E-12
Mercury, divalent	8.55E-08	3.18E-08	1.58E-08	1.58E-08	3.79E-10	1.63E-07	5.86E-08	3.14E-08	3.14E-08	7.51E-10
1,4-Dichlorobenzene	4.82E-12	3.39E-12	2.84E-13	1.14E-10	7.77E-12	4.83E-12	3.40E-12	2.85E-13	1.14E-10	7.79E-12
Acenaphthene	5.44E-11	2.59E-11	8.00E-13	3.20E-10	2.52E-11	5.45E-11	2.60E-11	8.06E-13	3.23E-10	2.54E-11
Acenaphthylene	2.05E-09	9.62E-10	3.68E-12	1.47E-09	2.42E-10	2.08E-09	9.74E-10	4.29E-12	1.72E-09	2.58E-10
Anthracene	5.16E-11	2.16E-11	7.81E-13	3.12E-10	2.21E-11	5.22E-11	2.19E-11	7.97E-13	3.19E-10	2.26E-11
Arsenic	1.19E-07	5.35E-09	NA	NA	NA	1.19E-07	5.36E-09	NA	NA	NA
Benzene	3.38E-13	2.43E-13	1.58E-14	6.26E-12	5.62E-13	3.38E-13	2.43E-13	1.56E-14	6.26E-12	5.62E-13
Benzo(a)anthracene	2.32E-08	9.30E-09	2.77E-10	1.11E-07	8.08E-09	2.41E-08	9.60E-09	3.02E-10	1.21E-07	8.73E-09
Benzo(a)pyrene	5.23E-08	2.42E-08	1.25E-10	5.01E-08	6.63E-09	5.26E-08	2.43E-08	1.34E-10	5.38E-08	6.87E-09
Benzo(b)fluoranthene	8.56E-08	3.66E-08	6.98E-10	2.81E-07	2.27E-08	8.76E-08	3.72E-08	7.57E-10	3.04E-07	2.42E-08
Benzo(k)fluoranthene	2.37E-07	1.06E-07	1.12E-09	4.48E-07	4.32E-08	2.49E-07	1.10E-07	1.47E-09	5.90E-07	5.24E-08
Benzo(g,h,i)perylene	1.60E-08	7.49E-09	3.28E-11	1.31E-08	2.04E-09	1.63E-08	7.59E-09	3.83E-11	1.54E-08	2.19E-09
Beryllium	4.63E-09	5.58E-12	NA	NA	NA	5.22E-09	6.07E-12	NA	NA	NA
Cadmium	2.67E-10	2.19E-11	9.16E-10	2.16E-11	4.75E-11	2.76E-10	2.29E-11	1.29E-09	3.03E-11	6.30E-11
Chromium	1.33E-06	4.49E-07	NA	NA	NA	1.92E-06	5.90E-07	NA	NA	NA
Chromium VI	2.21E-07	9.05E-08	NA	NA	NA	2.21E-07	9.05E-08	NA	NA	NA
Chrysene	3.20E-08	1.37E-08	2.46E-10	9.83E-08	8.11E-09	3.32E-08	1.41E-08	2.80E-10	1.12E-07	8.97E-09
Dibenz(a,h)anthracene	1.13E-06	5.36E-07	1.18E-09	4.71E-07	1.15E-07	1.14E-06	5.37E-07	1.34E-09	5.36E-07	1.20E-07
Fluoranthene	2.75E-09	1.02E-09	4.78E-11	1.91E-08	1.27E-09	2.85E-09	1.05E-09	4.96E-11	1.99E-08	1.32E-09
Fluorene	4.54E-10	2.12E-10	9.04E-13	3.62E-10	5.24E-11	4.55E-10	2.13E-10	9.10E-13	3.64E-10	5.26E-11
Formaldehyde	3.28E-14	2.36E-14	1.51E-15	6.07E-13	5.45E-14	3.28E-14	2.36E-14	1.51E-15	6.07E-13	5.45E-14
Indeno(1,2,3-cd)pyrene	1.05E-05	4.99E-06	2.83E-09	1.13E-06	9.25E-07	1.05E-05	5.00E-06	3.13E-09	1.25E-06	9.33E-07
Lead	2.44E-09	2.76E-09	NA	NA	5.76E-10	2.91E-09	3.21E-09	NA	NA	8.29E-10
Naphthalene	6.76E-11	4.08E-11	1.94E-12	7.79E-10	6.87E-11	6.78E-11	4.09E-11	1.95E-12	7.82E-10	6.90E-11
Nickel	3.34E-04	8.27E-05	NA	NA	NA	3.35E-04	8.31E-05	NA	NA	NA
Phenanthrene	3.22E-12	2.17E-12	1.42E-13	5.69E-11	5.03E-12	3.23E-12	2.17E-12	1.43E-13	5.72E-11	5.05E-12
Pyrene	3.45E-08	1.62E-08	6.16E-11	2.47E-08	4.06E-09	3.50E-08	1.64E-08	7.20E-11	2.89E-08	4.34E-09
Toluene	1.70E-11	1.21E-11	7.76E-13	3.10E-10	2.78E-11	1.70E-11	1.21E-11	7.76E-13	3.10E-10	2.78E-11
Vanadium	5.85E-04	6.97E-04	NA	NA	NA	5.86E-04	6.97E-04	NA	NA	NA

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - No biotransfer factor is available for this compound for this medium.

Table 4-17

Exposure Point Concentrations in Animal Products Under Natural Gas High Load Conditions
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	For Evaluating Cancer Risk (mg/kg)					For Evaluating Non-Cancer Effects (mg/kg)				
	Beef	Milk	Chicken	Eggs	Pork	Beef	Milk	Chicken	Eggs	Pork
Mercury, elemental	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC
Mercury, methyl	6.89E-12	3.67E-12	5.55E-13	5.55E-13	1.44E-14	9.37E-12	4.53E-12	1.06E-12	1.06E-12	2.63E-14
Mercury, divalent	1.09E-09	4.11E-10	1.99E-10	1.99E-10	4.77E-12	2.07E-09	7.49E-10	3.95E-10	3.95E-10	9.46E-12
1,4-Dichlorobenzene	2.75E-11	1.93E-11	1.62E-12	6.47E-10	4.43E-11	2.75E-11	1.94E-11	1.62E-12	6.49E-10	4.44E-11
Acenaphthene	1.69E-12	8.05E-13	2.49E-14	9.96E-12	7.83E-13	1.69E-12	8.08E-13	2.51E-14	1.00E-11	7.90E-13
Acenaphthylene	4.12E-09	1.90E-09	1.19E-11	4.78E-09	5.68E-10	4.22E-09	1.94E-09	1.39E-11	5.59E-09	6.22E-10
Anthracene	3.69E-11	1.55E-11	5.59E-13	2.24E-10	1.58E-11	3.73E-11	1.57E-11	5.71E-13	2.28E-10	1.62E-11
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	2.85E-13	2.05E-13	1.32E-14	5.28E-12	4.74E-13	2.85E-13	2.05E-13	1.32E-14	5.28E-12	4.74E-13
Benzo(a)anthracene	3.46E-09	1.36E-09	4.64E-11	1.86E-08	1.31E-09	3.62E-09	1.41E-09	5.05E-11	2.02E-08	1.42E-09
Benzo(a)pyrene	1.78E-08	8.15E-09	5.62E-11	2.25E-08	2.48E-09	1.79E-08	8.20E-09	6.04E-11	2.42E-08	2.58E-09
Benzo(b)fluoranthene	1.44E-08	6.00E-09	1.44E-10	5.77E-08	4.38E-09	1.48E-08	6.12E-09	1.56E-10	6.26E-08	4.69E-09
Benzo(k)fluoranthene	4.40E-08	1.92E-08	2.84E-10	1.14E-07	9.61E-09	4.70E-08	2.01E-08	3.75E-10	1.50E-07	1.19E-08
Benzo(g,h,i)perylene	2.05E-09	9.36E-10	7.96E-12	3.19E-09	3.43E-10	2.12E-09	9.61E-10	9.31E-12	3.73E-09	3.79E-10
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	7.43E-09	3.10E-09	7.12E-11	2.84E-08	2.18E-09	7.80E-09	3.22E-09	8.09E-11	3.23E-08	2.43E-09
Dibenzo(a,h)anthracene	2.12E-07	9.88E-08	3.77E-10	1.51E-07	2.44E-08	2.13E-07	9.93E-08	4.29E-10	1.72E-07	2.57E-08
Fluoranthene	6.01E-10	2.20E-10	1.07E-11	4.30E-09	2.84E-10	6.24E-10	2.29E-10	1.12E-11	4.48E-09	2.96E-10
Fluorene	8.18E-11	3.79E-11	2.09E-13	8.37E-11	1.00E-11	8.19E-11	3.80E-11	2.10E-13	8.43E-11	1.00E-11
Formaldehyde	3.47E-13	2.50E-13	1.60E-14	6.42E-12	5.77E-13	3.47E-13	2.50E-13	1.60E-14	6.42E-12	5.77E-13
Indeno(1,2,3-cd)pyrene	2.00E-06	9.50E-07	1.06E-09	4.24E-07	1.86E-07	2.00E-06	9.51E-07	1.17E-09	4.69E-07	1.88E-07
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	3.90E-11	2.35E-11	1.12E-12	4.50E-10	3.97E-11	3.91E-11	2.36E-11	1.13E-12	4.52E-10	3.99E-11
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	1.90E-12	1.28E-12	8.40E-14	3.36E-11	2.97E-12	1.91E-12	1.28E-12	8.43E-14	3.38E-11	2.98E-12
Pyrene	1.14E-08	5.28E-09	3.31E-11	1.33E-08	1.58E-09	1.17E-08	5.38E-09	3.87E-11	1.55E-08	1.73E-09
Toluene	1.82E-12	1.30E-12	8.33E-14	3.34E-11	2.99E-12	1.82E-12	1.30E-12	8.34E-14	3.34E-11	2.99E-12
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted in this operating scenario.

Table 4-18
Exposure Parameter Values
Human Health Risk Assessment
Coleston Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

	Resident Adult		Source	Resident Child		Source	Farmer Adult		Source	Farmer Child		Source	Fisher Adult		Source	Fisher Child		Source
Exposure frequency (d/y)	350	(c)		350	(c)		350	(c)		350	(c)		350	(c)		350	(c)	
Exposure duration (y)	30	(c)		6	(c)		30	(c)		6	(c)		30	(c)		6	(c)	
Body weight (kg)	70	(a)		13	(a)		70	(a)		13	(a)		70	(a)		13	(a)	
Averaging time for carcinogens (d)	25550	(c)		25550	(c)		25550	(c)		25550	(c)		25550	(c)		25550	(c)	
Averaging time for noncarcinogens (d)	10950	(c)		2190	(c)		10950	(c)		2190	(c)		10950	(c)		2190	(c)	
Soil ingestion rate (mg/d)	20	(b)		200	(b)		20	(b)		200	(b)		20	(b)		200	(b)	
Fraction of soil ingested that is from impacted area (unitless)	1	(c)		1	(c)		1	(c)		1	(c)		1	(c)		1	(c)	
Conversion factor (kg/mg)	0.000001	(c)		0.000001	(c)		0.000001	(c)		0.000001	(c)		0.000001	(c)		0.000001	(c)	
Drinking water ingestion rate (L/d)	1.5	(a)		0.8	(a)		1.5	(a)		0.8	(a)		1.5	(a)		0.8	(a)	
Inhalation rate (m3/d)	23	(a)		5	(a)		23	(a)		5	(a)		23	(a)		5	(a)	
Fraction of air from affected area (unitless)	1	(c)		1	(c)		1	(c)		1	(c)		1	(c)		1	(c)	
Conversion factor (ug/mg)	0.001	(c)		0.001	(c)		0.001	(c)		0.001	(c)		0.001	(c)		0.001	(c)	
Produce (above ground) ingestion rate (g/d)	60.9	(c)		18.85	(c)		60.9	(c)		18.85	(c)		60.9	(c)		18.85	(c)	
Produce (root) ingestion rate (g/d)	9.8	(c)		3.3	(c)		9.8	(c)		3.3	(c)		9.8	(c)		3.3	(c)	
Fraction of produce that is from affected area (unitless)	0.25	(c)		0.25	(c)		1	(c)		1	(c)		0.25	(c)		0.25	(c)	
Conversion factor (g/g)	0.001	(c)		0.001	(c)		0.001	(c)		0.001	(c)		0.001	(c)		0.001	(c)	
Fish ingestion rate (g/d)													81.9	(c)		11.385	(c)	
Fraction of fish from affected area (unitless)													1	(c)		1	(c)	
Conversion factor (kg/g)	0.001	(c)		0.001	(c)		0.001	(c)		0.001	(c)		0.001	(c)		0.001	(c)	
Beef ingestion rate (g/d)							79.8	(c)		7.65	(c)							
Fraction of beef that is from affected area (unitless)							1	(c)		1	(c)							
Conversion factor (kg/g)							0.001	(c)		0.001	(c)							
Milk ingestion rate (g/d)							670	(a)		283	(a)							
Fraction of milk that is from affected area (unitless)							1	(c)		1	(c)							
Conversion factor (kg/g)							0.001	(c)		0.001	(c)							
Poultry ingestion rate (g/d)							42.7	(c)		6.375	(c)							
Fraction of poultry from affected area (unitless)							1	(c)		1	(c)							
Conversion factor (kg/g)							0.001	(c)		0.001	(c)							
Egg ingestion rate (g/d)							43.4	(c)		6.57	(c)							
Fraction of eggs from affected area (unitless)							1	(c)		1	(c)							
Conversion factor (kg/g)							0.001	(c)		0.001	(c)							
Pork ingestion rate (g/d)							37.1	(c)		5.97	(c)							
Fraction of pork from affected area (unitless)							1	(c)		1	(c)							
Conversion factor (kg/g)							0.001	(c)		0.001	(c)							

Notes:

(a) Health Canada, 1994. Human Health Risk Assessment for Priority Substances.

(b) Atlantic PIRI, 1999. Atlantic RBCA Reference Documentation for Petroleum Impacted Sites. Version 1.0.

(c) U.S. EPA, 1998.

5.0 RISK CHARACTERIZATION

Risk characterization is the step in the risk assessment process that combines the results of the exposure assessment and the toxicity assessment for each compound of concern (CPC) in order to estimate the potential for carcinogenic and noncarcinogenic human health effects from chronic exposure to that compound. This section summarizes the results of the risk characterization for each receptor evaluated in the risk assessment for each of the three operating scenarios evaluated in the risk assessment. The noncarcinogenic risk characterization process is discussed in Section 5.1, and Section 5.2 presents the carcinogenic risk characterization process.

Section 5.3 presents the risk characterization results for the Existing Operation - Fuel Oil #6 - High Load Scenario. The risk characterization results for the Refurbishment Project - Orimulsion® - High Load Scenario are presented in Section 5.4. The risk characterization results for the Natural Gas - High Load Scenario are discussed in Section 5.5.

Section 5.6 presents a comparison of the risk assessments of the three operating scenarios, and Section 7 discusses a summary and conclusions of the risk characterization of non-criteria pollutants.

Sections 5.8 through 5.11 present a qualitative evaluation of potential respiratory effects from exposure to facility emissions, including discussions of air quality in Saint John, health status of residents of Saint John and the province of New Brunswick, cardiorespiratory health effects, and the prevalence and causes of asthma and other respiratory diseases.

A discussion of susceptible populations is presented in Section 5.12, and Section 5.13 includes a comparison of impacts of the CCGS to background conditions. Section 5.14 presents the conclusions about effects of the Coleson Cove Generating Station on cardiorespiratory health outcomes. Section 5.15 presents a risk characterization of criteria pollutants.

5.1 Noncarcinogenic Risk Characterization

The potential for exposures to CPCs at the CCGS to result in adverse noncarcinogenic health effects is estimated for each combination of compound/exposure pathway/receptor by comparing the Chronic Average Daily Dose (CADD) for that compound/exposure pathway/receptor (derived in Section 4.0) with the Tolerable Daily Intake (TDI) for that compound (presented in Section 3.0). The resulting ratio, which is unitless, is known as the Hazard Quotient (HQ) for that compound. The HQ is calculated using the following formula:

$$A = \frac{B}{C}$$

where:

A = Hazard Quotient (unitless);
B = Chronic Average Daily Dose (mg/kg-day); and
C = Tolerable Daily Intake (mg/kg-day).

When the Hazard Quotient for a given compound does not exceed 1, the TDI has not been exceeded, and no adverse noncarcinogenic health effects are expected to occur as a result of that receptor's potential exposure to that compound via that exposure pathway. The regulatory benchmark of 1 is widely used by Canada and the United States. The HQs for each pathway are summed to yield the Hazard Index (HI) for that compound for that receptor. A Total Hazard Index (HI) is then calculated for each receptor for each toxic endpoint by summing the chemical-specific HIs for all chemicals that have the same toxic endpoint. An endpoint-specific HI for a chemical that does not exceed 1 for a given receptor indicates that no adverse noncarcinogenic health effects are expected to occur as a result of that receptor's potential exposure to that chemical in the environmental media potentially affected by the facility. As a screening measure, all chemical-specific HIs are summed regardless of toxic endpoint. If this value does not exceed 1, then it is not necessary to perform the endpoint-specific analysis.

Hazard Quotients calculated for each compound/exposure pathway/receptor are presented in Appendix B. The HIs associated with each receptor's exposure to the compounds in emissions from the CCGS under each of the three sets of operating conditions are discussed in Sections 5.3 through 5.5.

5.2 Carcinogenic Risk Characterization

The purpose of carcinogenic risk characterization is to estimate the likelihood, over and above the background cancer rate, that a receptor will develop cancer in his or her lifetime as a result of facility-related exposures to CPCs in various environmental media. This likelihood is a function of the dose of a compound and the Cancer Slope Factor (CSF) for that compound. The relationship between the Excess Lifetime Cancer Risk (ELCR) and the estimated Lifetime Average Daily Dose (LADD) of a compound may be expressed as:

$$A = 1 - e^{-BC}$$

where:

A = Excess Lifetime Cancer Risk (unitless);
B = Cancer Slope Factor (1/(mg/kg-day)); and
C = Lifetime Average Daily Dose (mg/kg-day).

When the product of the CSF and the LADD is much greater than 1, the ELCR approaches 1 (i.e., 100% probability). When the product is less than 0.01 (1×10^{-2}), the equation can be closely approximated by:

$$A = B \times C$$

where:

A = Excess Lifetime Cancer Risk (unitless);
B = Cancer Slope Factor (1/(mg/kg-day)); and
C = Lifetime Average Daily Dose (mg/kg-day).

For each combination of compound/exposure pathway/receptor, the product of the CSF and the LADD is unitless, and provides an estimate of the potential carcinogenic risk associated with that receptor's exposure to that compound via that pathway. ELCRs are calculated for each potentially carcinogenic compound. For each receptor, the ELCRs for each compound is calculated by summing the potential risks derived for each pathway. A Total Excess Lifetime Cancer Risk for the receptor is then calculated by summing the compound-specific ELCRs. In accordance with risk assessment policy for emissions from combustion sources, if the ELCR for a receptor is less than a target risk of 1×10^{-5} , then the risk assessment will conclude that the ELCR for that compound falls within the acceptable range of potential risks.

Excess Lifetime Cancer Risks calculated for each compound/exposure pathway/receptor are presented in Appendix B. The ELCRs associated with each receptor's exposure to the compounds in emissions from the CCGS under each of the three sets of operating conditions are discussed in Sections 5.3 through 5.5.

5.3 Existing Operation - Fuel Oil #6 - High Load Scenario

In the Existing Operation - Fuel Oil #6 - High Load Scenario, the CCGS is assumed to use heavy fuel oil as a source of fuel, operating 24 hours per day, 365 days per year, for 30 years. Potential receptors evaluated in the Existing Operation - Fuel Oil #6 - High Load Scenario include a resident living at the maximum hypothetical residential area downwind of the facility, a resident living in Saint John, a recreational fisher living at the maximum hypothetical residential area downwind of the facility, a recreational fisher living in Saint John, and a farmer living at a farm on the peninsula northeast of the facility.

5.3.1 Resident at the Maximum Hypothetical Residence

The hazard indices associated with the maximum hypothetical resident's exposure to compounds emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-1. The Total HI is 0.2 for the child resident and 0.08 for the adult resident. The compound-specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the closest resident's exposure to CPCs emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-2. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 7×10^{-8} for the child resident and 1×10^{-7} for the adult resident, which are also less than the target risk of 1×10^{-5} .

5.3.2 Resident Living in Saint John

The hazard indices associated with the Saint John resident's exposure to compounds emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-3. The Total HI is 0.1 for the child resident and 0.04 for the adult resident. The compound-specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the Saint John resident's exposure to CPCs emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-4. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 7×10^{-8} for the child resident and 2×10^{-7} for the adult resident, which are also less than the target risk of 1×10^{-5} .

5.3.3 Recreational Fisher Living at the Maximum Hypothetical Residence

The hazard indices associated with the exposures of the recreational fisher at the maximum hypothetical residence to compounds emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-5. The compound-specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, indicating that, even when potential exposure to these compounds from other sources unrelated to the CCGS, noncarcinogenic health effects are not likely to occur for this receptor.

Because the total hazard index for the child recreational fisher regardless of toxicity endpoint is the highest of all receptors, an endpoint-specific analysis was performed.

The sum of the individual HQs for the fisher child is 0.31. The following summarizes the individual chemical HQs and their toxic endpoints.

Arsenic	0.002	Skin effects
Lead	0.015	Nervous system effects
Nickel	0.020	Changes in body weight and organ/body weight ratios
Mercury	0.090	Nervous system effects, immune system effects
Vanadium	0.181	No adverse effects seen at highest dose (hair effects seen)

As can be seen, only lead and mercury have a toxicity endpoint in common. Thus, it is appropriate to sum the HQs for lead and mercury to derive a Nervous System-Specific Hazard Index of 0.105. No other CPCs should be summed.

Thus the toxicity endpoint-specific HIs are:

Skin Effects	0.002
Nervous System Effects	0.105
Body Weight Effects	0.020
Immune System Effects	0.090
Hair Effects	0.181

It is interesting that the highest HI is seen with vanadium for which a TDI was derived from a study in which no adverse effects were noted. The researchers did note that cystein levels were higher in the hair of dosed rodents than in control rodents, but this was not identified as an adverse effect. Adverse effects were not seen in either of two dose groups. Nonetheless, the U.S. EPA defined the lower of the two doses as a No Observed Adverse Effect Level (NOAEL) for the purposes of defining a Reference Dose (equivalent to a TDI) and U.S. EPA defined the toxic endpoint as effects on hair. This dose was divided by 10 for an interspecies extrapolation uncertainty factor and by 10 to protect sensitive human sub-populations. Had the EPA used the higher of the two doses as the NOAEL for derivation of the reference dose which would conform to EPA guidance, the HI for vanadium for the fisher child would have been 0.018.

In conclusion, all individual chemical HIs are less than 0.18, and all toxicity endpoint- specific HIs are less than 0.18. Thus, the margin of safety for this receptor in addition to the margin of safety of 100-fold or more, which is inherent in the derivation of the TDIs, is an extra factor of 6.

The ELCRs associated with the maximum hypothetical residence recreational fisher's exposure to CPCs emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-6. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 8×10^{-8} for the child fisher and 2×10^{-7} for the adult fisher, which are also less than the target risk of 1×10^{-5} .

5.3.4 Recreational Fisher Living in Saint John

The hazard indices associated with the Saint John recreational fisher's exposure to compounds emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-7. The Total HI is 0.2 for the child fisher and 0.2 for the adult fisher. The compound-

specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the Saint John recreational fisher's exposure to CPCs emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-8. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 8×10^{-8} for the child fisher and 3×10^{-7} for the adult fisher, which are also less than the target risk of 1×10^{-5} .

5.3.5 Farmer

The hazard indices associated with the farmer's exposure to compounds emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-9. The Total HI is 0.1 for the child farmer and 0.04 for the adult farmer. The compound-specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the farmer's exposure to CPCs emitted from the CCGS in the Existing Operation - Fuel Oil #6 - High Load Scenario are shown in Table 5-10. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 5×10^{-8} for the child farmer and 1×10^{-7} for the adult farmer, which are also less than the target risk of 1×10^{-5} .

5.4 Refurbishment Project - Orimulsion® - High Load Scenario

In the Refurbishment Project - Orimulsion® - High Load Scenario, the CCGS is assumed to use Orimulsion® as a source of fuel, with the facility operating 24 hours per day, 365 days per year, for 30 years. Potential receptors evaluated in the Refurbishment Project - Orimulsion® - High Load Scenario include a resident living at the maximum hypothetical residence downwind of the facility, a resident living in Saint John, a recreational fisher living at the maximum hypothetical residence downwind of the facility, a recreational fisher living in Saint John, and a farmer living at a farm on the peninsula northeast of the facility.

5.4.1 Resident Living at Maximum Hypothetical Residence

The hazard indices associated with the maximum hypothetical resident's exposure to compounds emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-11. The total HI is 0.02 for the child resident and 0.009 for the adult resident. The compound-specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic

adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the closest resident's exposure to CPCs emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-12. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 7×10^{-9} for the child resident and 1×10^{-8} for the adult resident, which are also less than the target risk of 1×10^{-5} .

5.4.2 Resident Living in Saint John

The hazard indices associated with the Saint John resident's exposure to compounds emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-13. The Total HI is 0.01 for the child resident and 0.007 for the adult resident. The compound-specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the Saint John resident's exposure to CPCs emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-14. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 1×10^{-8} for the child resident and 5×10^{-8} for the adult resident, which are also less than the target risk of 1×10^{-5} .

5.4.3 Recreational Fisher Living at the Maximum Hypothetical Residence

The hazard indices associated with the exposures of the recreational fisher at the maximum hypothetical residence to compounds emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-15. The Total HI is 0.04 for the child fisher and 0.03 for the adult fisher. The compound-specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the maximum hypothetical residence recreational fisher's exposure to CPCs emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-16. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 8×10^{-9} for the child fisher and 2×10^{-8} for the adult fisher, which are also less than the target risk of 1×10^{-5} .

5.4.4 Recreational Fisher Living in Saint John

The hazard indices associated with the Saint John recreational fisher's exposure to compounds emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-17. The Total HI is 0.03 for the child fisher and 0.03 for the adult fisher. The compound-specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the Saint John recreational fisher's exposure to CPCs emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-18. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 2×10^{-8} for the child fisher and 6×10^{-8} for the adult fisher, which are also less than the target risk of 1×10^{-5} .

5.4.5 Farmer

The hazard indices associated with the farmer's exposure to compounds emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-19. The Total HI is 0.01 for the child farmer and 0.005 for the adult farmer. The compound-specific hazard indices (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the farmer's exposure to CPCs emitted from the CCGS in the Refurbishment Project - Orimulsion® - High Load Scenario are shown in Table 5-20. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 8×10^{-9} for the child farmer and 3×10^{-8} for the adult farmer, which are also less than the target risk of 1×10^{-5} .

5.5 Natural Gas - High Load Scenario

The risk assessment includes an evaluation of potential risks assuming that the facility is configured to use natural gas as a source of fuel. Potential receptors evaluated under comparison conditions include a resident living at the maximum hypothetical residence downwind of the facility, a resident living in Saint John, a recreational fisher living at the maximum hypothetical residence downwind of the facility, a recreational fisher living in Saint John, and a farmer living at a farm on the peninsula northeast of the facility.

5.5.1 Resident Living at the Maximum Hypothetical Residence

The hazard indices associated with the maximum hypothetical resident's exposure to compounds emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-21. The Total HI is 5×10^{-6} for the child resident and 1×10^{-6} for the adult resident. The compound-specific HIs (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the closest resident's exposure to CPCs emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-22. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 2×10^{-10} for the child resident and 3×10^{-10} for the adult resident, which are also less than the target risk of 1×10^{-5} .

5.5.2 Resident Living in Saint John

The hazard indices associated with the Saint John resident's exposure to compounds emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-23. The Total HI is 1×10^{-6} for the child resident and 5×10^{-7} for the adult resident. The compound-specific HIs (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the Saint John resident's exposure to CPCs emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-24. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 8×10^{-11} for the child resident and 3×10^{-10} for the adult resident, which are also less than the target risk of 1×10^{-5} .

5.5.3 Recreational Fisher Living at the Maximum Hypothetical Residence

The hazard indices associated with the exposures of the recreational fisher at the closest residential area to compounds emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-25. The Total HI is 3×10^{-4} for the child fisher and 3×10^{-4} for the adult fisher. The compound-specific HIs (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the closest recreational fisher's exposure to CPCs emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-26. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds.

The total ELCR (which combines all chemicals and all pathways) is 6×10^{-10} for the child fisher and 3×10^{-9} for the adult fisher, which are also less than the target risk of 1×10^{-5} .

5.5.4 Recreational Fisher Living in Saint John

The hazard indices associated with the Saint John recreational fisher's exposure to compounds emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-27. The Total HI is 3×10^{-4} for the child fisher and 3×10^{-4} for the adult fisher. The compound-specific HIs (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the Saint John recreational fisher's exposure to CPCs emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-28. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 4×10^{-10} for the child fisher and 3×10^{-9} for the adult fisher, which are also less than the target risk of 1×10^{-5} .

5.5.5 Farmer

The hazard indices associated with the farmer's exposure to compounds emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-29. The Total HI is 8×10^{-7} for the child farmer and 4×10^{-7} for the adult farmer. The compound-specific HIs (as a sum of all the pathways) are less than 1 for all compounds. These results indicate that the Facility emissions will not result in noncarcinogenic adverse health effects for this receptor. The total HI summed over all chemicals regardless of toxicity endpoint is also less than 1.0, again indicating that noncarcinogenic health effects are not likely to occur for this receptor.

The ELCRs associated with the farmer's exposure to CPCs emitted from the CCGS in the Natural Gas - High Load Scenario are shown in Table 5-30. The compound-specific ELCRs (as a sum of all the pathways) are less than the target risk of 1×10^{-5} for all compounds. The total ELCR (which combines all chemicals and all pathways) is 5×10^{-11} for the child farmer and 2×10^{-10} for the adult farmer, which are also less than the target risk of 1×10^{-5} .

5.6 Comparison of Three Operating Conditions

The risk assessment estimated potential risks associated with exposures of several types of receptors to emissions from the CCGS under three sets of operating conditions:

- Existing Operation - Fuel Oil #6 - High Load Scenario, in which the facility is assumed to continue to use Heavy Fuel Oil and operates continuously at maximum capacity for 30 years;
- Refurbishment Project - Orimulsion® - High Load Scenario, in which the facility is assumed to use Orimulsion® and operates continuously at maximum capacity for 30 years; and

- Natural Gas - High Load Scenario, which assumes that the facility uses natural gas and operates continuously at maximum capacity for 30 years.

The three cases differ with respect to the configuration of the stack and the types of emission control equipment assumed to be in use. Accordingly, the compounds that may be released in stack emissions, as well as the concentrations of those compounds, differ among the three sets of conditions. Potential carcinogenic and noncarcinogenic risks are estimated for each set of operating conditions.

For each set of operating conditions, the estimated carcinogenic and noncarcinogenic risks for each compound are less than the target risks for all receptors, indicating that the estimated risks fall within the range of acceptable risks and do not exceed any regulatory levels of concern.

However, although all estimated risks are within acceptable ranges, the estimated risks for each set of operating conditions differ from each other. Potential risks associated with the Existing Operation - Fuel Oil #6 - High Load Scenario are somewhat higher than potential risks associated with the Refurbishment Project - Orimulsion® - High Load Scenario. For example, the receptor with the highest potential risks (for both sets of operating conditions) is the adult fisher. For this receptor, the highest potential noncarcinogenic risks are associated with consumption of fish that are assumed to have taken up mercury from facility emissions. In the Existing Operation - Fuel Oil #6 - High Load Scenario, the HQ due to fish consumption is 0.090 while the HQ for fish consumption in the Refurbishment Project - Orimulsion® - High Load Scenario is 0.016. Although both HQs are less than 1, the HQ in the Refurbishment Project - Orimulsion® - High Load Scenario is six-fold lower than that in the Existing Operation - Fuel Oil #6 - High Load Scenario.

5.7 Summary and Conclusions of Risk Characterization

The results of the noncarcinogenic risk characterization indicate that both total and compound-specific hazard indices for the various receptors are less than 1 for all three sets of operating conditions. Therefore, no noncarcinogenic adverse health effects are expected to occur for any receptors as a result of potential exposure to emissions from the CCGS under all three sets of operating conditions.

The results of the carcinogenic risk characterization indicate that both total and compound-specific ELCRs are less than 1×10^{-5} under all three sets of operating conditions. Therefore, the total ELCRs for all sets of operating conditions fall within acceptable risk ranges and do not exceed any regulatory levels of concern.

Among the three sets of operating conditions evaluated in the risk assessment, potential risks are estimated to be highest (although still within acceptable ranges) for the Existing Operation - Fuel Oil #6 - High Load Scenario. Potential risks associated with the Refurbishment Project - Orimulsion® - High Load Scenario are somewhat lower than the potential risks associated with Existing Operation - Fuel Oil #6 - High Load Scenario. Potential risks associated with the Natural Gas - High Load Scenario, assuming the facility uses natural gas as a fuel source, were lower

than the potential risks associated with the Refurbishment Project - Orimulsion® - High Load Scenario.

However, cancer risks estimated by this risk assessment for all three fueling scenarios are all so low that they are immeasurable and indistinguishable from each other. Although the numerical estimates can be shown in the various tables to be different numbers, some of which are lower than others, they are so low that any differences between have limited, if any, significance. According to the Atlantic Health Sciences Corporation (2001), the population of the City of Saint John in 2000 was 72,269. The estimated excess lifetime cancer risk from this risk assessment that would be equivalent to one additional case of cancer in Saint John over a year is 4.1×10^{-4} . The estimated excess lifetime cancer risk from this risk assessment that would be equivalent to one additional case of cancer in Saint John over the course of a 30 year period is 1.4×10^{-5} . Clearly, any estimated risk level that is less than 4.1×10^{-4} is not measurable over a year and any estimated risk level that is less than 1.4×10^{-5} is not measurable over 30 years.

According to Atlantic Health Sciences Corporation (2001), cancer incidence rates for the City of Saint John were 218 cases (total cancers) in 2001 in males and 195 cases (total cancers) in females in 2001. The total number of cases was thus 413 out of a population of 72,269. If the estimated excess lifetime cancer risk from this risk assessment for any fueling scenario was 4.1×10^{-4} or higher, then one would predict that this number of cancer cases would increase from 413 to 414 cases in the population in a year. However, no estimated lifetime cancer risk was higher than 3×10^{-7} . Thus, the predicted increase in cancer cases per year would be from 413 cases to 413.0007 cases. This increase in risk is not measurable.

Over the course of the assumed period of operation of 30 years, there will be more cases of cancer in Saint John due to all of the many causes of cancer. If it is assumed that the cancer incidence rate due to all sources remains constant and the population remains constant, one can predict that the number of cases of cancer over 30 years will be $413 \times 30 = 12,390$. If the estimated excess lifetime cancer risk from this risk assessment for any fueling scenario was 1.4×10^{-5} or higher, then one would predict that this number of cancer cases would increase from 12,390 to 12,391 cases in the population over the course of 30 years. Such a risk, although small, would be measurable. However, no estimated lifetime cancer risk was higher than 3×10^{-7} . Thus, the predicted increase in cancer cases over 30 years would be from 12,390 cases to 12,390.02 cases. This increase in risk is also not measurable.

In conclusion, it should be viewed as a general benefit to public health that the estimated lifetime cancer risk estimates for all receptors decrease with the proposed refurbishment project. For instance, the estimated excess lifetime cancer risk for the Saint John resident receptor drops by a factor of 4. However, this is a theoretical decrease in risk, because the estimated risk with the Existing Condition - Fuel Oil #6 - High Load Scenario is immeasurable and far below regulatory levels of concern. With the Refurbishment Project - Orimulsion® - High Load Scenario, the cancer risks are estimated to drop by 4-fold to a lower, but still immeasurable level that is also far below regulatory levels of concern. Similarly, with the Natural Gas - High Load Scenario, the cancer risks drop to even lower numbers, but the Orimulsion and natural gas scenarios should be

viewed as indistinguishable in risk, because neither is measurable and both are far below regulatory levels of concern.

Noncancer risks estimated by this risk assessment for all three fueling scenarios are all so low that they are indistinguishable from each other. Although the numerical estimates for Hazard Quotients and Hazard Indices can be shown in the various tables to be different numbers, some of which are lower than others, they are so low that any differences between them are irrelevant.

All Tolerable Daily Doses (TDI) and Reference Doses (RfDs), which are levels of a chemical to which a person can be exposed every day for their entire lifetime without experiencing an adverse effect on their health, are derived with many safety factors. Thus, a Hazard Index over 1.0 would exceed a regulatory level of concern, but it would not exceed a level at which adverse effects have ever been demonstrated in either animals or humans.

For instance, the arsenic HQ for the fisher child receptor at the maximum hypothetical residential location for the heavy fuel oil, high load scenario is 0.002. This value is 500 times lower than the TDI. However, the TDI was derived from a no effect level in humans divided by an additional safety factor of 3. The estimated dose for this receptor is, in fact, 25,000 times lower than the lowest dose level that was shown to cause adverse effects on the skin of humans exposed to arsenic in their drinking water.

Similarly, the vanadium HQ for this receptor is 0.18. This value is 6 times lower than the TDI. However, the TDI was defined as 100 times lower than the lowest dose in an animal experiment where no adverse effects were seen, even in the animals given a 10-fold higher dose of vanadium. The estimated dose to the fisher child at this location is, in fact, over 5,000 times lower than the dose level in the animal experiment where no adverse effects were seen.

As well, the mercury HQ for the child fisher receptor at this location is 0.09. This value is 11 times lower than the TDI. However, the TDI was defined as 1,000 times lower than the lowest dose in an animal experiment that resulted in adverse effects on the animals. The estimated dose to the fisher child is, in fact, over 10,000 times lower than the dose level in the animal experiment that caused adverse effects.

The TDIs and RfDs are defined by regulatory agencies to be intentionally very health-protective. The agencies assume that all dose levels within 10 fold of an effect level could also have adverse effects. They assume that humans are more sensitive to the chemical compared to animals in all cases, by a factor of 10. They always assume that some sensitive human subpopulations are 10-fold more sensitive than normal humans. All of these assumptions are made so that estimated doses of chemicals from risk assessments that yield HQ estimates less than 1.0 are certain to be totally devoid of risk.

In addition, if the hazard quotient is less than 1.0, the additional margin of safety addresses the concern that is sometimes raised that people may be exposed to background levels of the chemical from other sources. In this case, the highest toxicity endpoint-specific hazard index is 0.18, which means that even if background exposures were five times higher than the facility-

related exposures, the regulatory level of concern hazard index of 1 would still not be exceeded. Thus, all hazard index estimates from this risk assessment are essentially indistinguishable. In all cases, there is a high degree of certainty that no adverse effects will occur.

For instance, the HI for all chemicals combined for the resident child assumed to live in Saint John for the Orimulsion®, high load scenario is 0.01. This is 100 times lower than the noncancer benchmark of HI=1.0. No endpoint specific HI exceeds 0.008, which is 125 times lower than the 1.0 benchmark. In this case, there is a high degree of certainty that no adverse effects will occur. For the same receptor, the natural gas, high load scenario gives a total HI summed over all chemicals of 1×10^{-6} , which is far lower than the benchmark of 1.0, but these two results are, in fact, indistinguishable given the manner in which TDIs are derived. Noncancer risk is inversely proportional to Hazard Index as the Hazard Index drops from high values down to the benchmark of 1.0. However, once the estimated Hazard Index drops to levels below the benchmark level of concern, the differences in the Hazard Index are no longer distinguishable from one another.

In conclusion, for both carcinogenic and noncarcinogenic risk, the risks for all three scenarios are low enough that they should be viewed as essentially indistinguishable from each other in terms of a direct comparison to regulatory decision-making criteria. Reducing risks to levels below regulatory levels of concern can be viewed as adding societal benefits, to a point, because reduction of carcinogenic and noncarcinogenic risks to levels beyond required regulatory levels can decrease the uncertainty that is inherent in the risk estimation process. Thus, if one scenario has an estimated risk that is ten-fold below the regulatory level of concern, there is a societal benefit to choosing a second scenario that has an estimated risk that is 100 times below the regulatory benchmark, because one has increased certainty that if important factors were inadvertently left out or underestimated, the true result is more likely to still be acceptable if the margin of safety is greater for the second case. However, this increase in certainty cannot be extended *ad infinitum*. At some point reasonable people who evaluate the conservativeness of the risk assessment process, which has many safety factors inherently built into it, will have to conclude that an *additional* safety factor is simply not necessary. By this logic, one can conclude that reducing cancer risk from 1×10^{-7} to 1×10^{-8} can be viewed as having a societal benefit given a regulatory level of concern of 1×10^{-5} , but reducing it to 1×10^{-10} provides no tangible benefit.

This is the situation with the three fueling scenarios. Reducing the estimated cancer risk from 0.03 cases in 100,000 for the Existing Condition - Fuel Oil #6 - High Load scenario to 0.006 cases in 100,000 for the Refurbishment Project - Orimulsion® - High Load Scenario can be viewed as conferring a societal benefit in terms of reducing any uncertainty inherent in the risk assessment. In the first case, the additional margin of safety (over and above the margins of safety that are built into the risk assessment process) that the regulatory goal was met was 33-fold. In the second case, the additional margin of safety is 166-fold. Further reducing the estimated cancer risk to 0.0003 cases in 100,000 for the Natural Gas - High Load Scenario may indeed increase the margin of safety further, but attaining this additional reduction in risks does not equate to a proportionally increased benefit, because the result is already so far beyond the regulatory goal.

5.8 Air Quality in Saint John

According to New Brunswick Health and Wellness (2002), the air quality in Saint John is good compared with many industrialized nations. Air pollutants of concern for Saint John have been identified as ground-level ozone, sulfate, and acidic particles. Respirable particles (PM_{10} and $PM_{2.5}$) are "low to moderate in the Saint John area compared with other areas in Canada. In addition, PM_{10} is influenced more strongly by local sources compared to $PM_{2.5}$."

Sulphur dioxide levels have been declining in Saint John since 1976. The mean annual concentrations for four monitoring stations in 1996 ranged from 5-7 ppb (Forest Hills, Champlain Heights, Customs Building, and Regional Center/Hillcrest).

According to Hughes (2002), vanadium and nickel levels in $PM_{2.5}$ particulate have decreased slightly in Saint John (Forest Hills monitoring station) from 1992 to 1999. Average levels were about 0.02 ug/m^3 for vanadium and 0.004 ug/m^3 for nickel in 1999. Chromium levels have also declined from 1992 to 1999. No chromium was detected in 1999 with a detection limit of approximately 0.0002 ug/m^3 . Lead shows a decline since 1992 and little change since 1996. Average levels were about 0.002 ug/m^3 for lead in 1993.

According to Environment Canada/Health Canada (2000), the mean concentration of PM_{10} from 1984-1995 for Saint John was 15 ug/m^3 . The range of mean concentrations over the same period throughout Canada was $11\text{-}42 \text{ ug/m}^3$. Out of 28 monitors, Saint John was equal or lower than 24 monitors in other cities. The upper 90th percentile concentration of PM_{10} from 1984-1995 for Saint John was 28 ug/m^3 . The range of upper 95th percentile concentrations over the same period throughout Canada was $19\text{-}74 \text{ ug/m}^3$. Out of 28 monitors, Saint John was equal or lower than 23 monitors in other cities. The mean concentration of PM_{10} from four SSI monitors in Saint John from 1988-1994 was 15 ug/m^3 . The range of mean concentrations of nine other SSI monitors in other cities was $18\text{-}28 \text{ ug/m}^3$. The mean upper 95th percentile concentration of PM_{10} from four SSI monitors in Saint John from 1988-1994 was 27 ug/m^3 . The range of mean concentrations of nine other SSI monitors in other cities was $28\text{-}49 \text{ ug/m}^3$. Thus, over the period 1984-1995, PM_{10} concentrations in Saint John were lower than in most other cities across Canada.

The mean concentration of $PM_{2.5}$ from 1984-1995 for Saint John was 8.5 ug/m^3 . The range of mean concentrations over the same period throughout Canada was $6.9\text{-}20.2 \text{ ug/m}^3$. Out of 28 monitors, Saint John was equal or lower than 24 monitors in other cities. The upper 90th percentile concentration of PM_{10} from 1984-1995 for Saint John was 16.2 ug/m^3 . The range of upper 95th percentile concentrations over the same period throughout Canada was $10.9\text{-}39 \text{ ug/m}^3$. Out of 28 monitors, Saint John was equal or lower than 23 monitors in other cities. Thus, over the period 1984-1995, $PM_{2.5}$ concentrations in Saint John were lower than in most other cities across Canada.

According to the National Air Pollution Surveillance (NAPS) Network (JWEL, 2002), the mean PM_{10} concentration at the Forest Hills station from 1995-2000 was 14 ug/m^3 . At the 110 Charlotte Street station the mean PM_{10} from 1995-2000 was 22 ug/m^3 . The mean of both stations was 18

ug/m³ over this period. The mean PM_{2.5} concentration over this same period was 9 ug/m³ at Forest Hills.

5.9 Health Status of Residents of Saint John and the Province of New Brunswick

Various summary health statistics for southwestern New Brunswick have been compiled in a report entitled *Health Status Report 2001* (Atlantic Health Sciences Corporation, 2001). These health statistics are presented as indicators of the health status of the population of southwestern New Brunswick.

In this report, southwestern New Brunswick is designated as New Brunswick Health Region 2, one of seven designated health regions in the province of New Brunswick. Health Region 2 is further subdivided into four areas: Saint John City, Saint John Area (Saint John Census Metropolitan Area, excluding Saint John City and including Hampstead Parish), Charlotte County Area, and Sussex Area. In addition to New Brunswick Health Region 2, health statistics are also provided for the province of New Brunswick, Canada, and the other six New Brunswick Health Regions.

The types of statistics extracted from *Health Status Report 2001* and reported here include the following: cancer incidence, cancer mortality, and respiratory disease data (including mortality and hospital discharge rates). It should be noted that, in most cases, the statistical significance of any rate differences between regions or areas has not been tested. Therefore, it is possible that nominal differences in rates could be artifacts of reporting and due to chance alone.

Table 5-31 contains cancer incidence rates (per 100,000 population) for various types of cancer sites for Canada, New Brunswick, Health Region 2, and the four Health Region 2 areas. The cancer incidence data for Canada and New Brunswick have been age-standardized (that is, have been adjusted to account for the effect of age differences in the population on the rate of cancer) while the incidence data for New Brunswick Region 2 and its four areas are crude incidence rates that have not been age-adjusted. Also, the data for Canada and New Brunswick reflect the population's experience for the year 2001 while for Health Region 2 and its sub-areas, the cases span 1989 through 1998 and represent an average for that period. For these reasons, care should be exercised in comparing the data sets, as noted in the report, itself.

Lung cancer has been of particular concern to area residents and therefore will be reported here. As seen in Table 5-31, the lung cancer incidence rates for Canada as a whole are 77 lung cancer cases per 100,000 males and 47 cases per 100,000 females. For New Brunswick as a whole, the rate of lung cancer is 95 cases per 100,000 males and 50 cases per 100,000 females. The unadjusted lung cancer incidence rates for Health Region 2 and its four areas are also reported in Table 5-31. The lung cancer incidence rates for Region 2 are 107 cases per 100,000 males and 59 cases per 100,000 females. For Saint John City, the lung cancer incidence rates are 139 per 100,000 males and 79 per 100,000 females. For Saint John Area, the rates are 63 per 100,000 males and 37 per 100,000 females. Thus, lung cancer incidence appears to be slightly elevated in New Brunswick compared to Canada and in Saint John City compared to the province as a

whole. However, incidence rates in the Saint John Area appear to be lower than Saint John City, the province, and the country as a whole.

The incidence rates for other cancers follow a similar pattern. In males, rates for all cancers combined, prostate, colorectal, bladder, kidney, oral, stomach, pancreas, and larynx cancer are elevated in the City of Saint John compared to the province and the country as a whole, but the rates of the same cancers are lower for the Saint John Area compared to the province and the country as a whole. In females, rates for all cancers combined, breast, colorectal, uterus, non-Hodgkin's lymphoma, ovary, pancreas, cervix, kidney, leukemia, bladder, oral, stomach, and multiple myeloma are elevated in the City of Saint John compared to the province and the country as a whole, but the rates of the same cancers are lower for the Saint John Area compared to the province and the country as a whole. Air pollution has not been demonstrated to be causally associated with all of these cancer sites, so this general increase in cancers in the City of Saint John must have another explanation.

Table 5-32 contains cancer mortality rates for Canada, New Brunswick, Health Region 2, and the four Health Region 2 areas. The same limitations exist for the cancer mortality data as for the incidence data. As seen in Table 5-32, the lung cancer mortality rates for Canada as a whole (for the year 2001) are 69 lung cancer deaths per 100,000 males and 37 deaths per 100,000 females. For New Brunswick as a whole, the rate of lung cancer deaths is 89 per 100,000 males and 32 per 100,000 females. The unadjusted lung cancer mortality rates for Health Region 2 are 89 deaths per 100,000 males and 48 deaths per 100,000 females. For Saint John City, the mortality rate from lung cancer is 115 per 100,000 males and 68 per 100,000 females. For Saint John Area, the rates are 49 per 100,000 males and 31 per 100,000 females. Thus, lung cancer mortality appears to be slightly elevated in New Brunswick compared to Canada for males and in Saint John City compared to the province as a whole for both males and females. However, mortality rates in the Saint John Area appear to be lower than Saint John City, the province, and the country as a whole.

The mortality rates for other cancers follow a similar pattern. In males, rates for prostate, colorectal, bladder, pancreas, and larynx cancer are elevated in the City of Saint John compared to the province and the country as a whole, but the rates of the same cancers are lower for the Saint John Area compared to the province and the country as a whole. In females, rates for breast, colorectal, uterus, non-Hodgkin's lymphoma, ovary, pancreas, cervix, kidney, leukemia, bladder, oral, and stomach are elevated in the City of Saint John compared to the province and the country as a whole, but the rates of the same cancers are lower for the Saint John Area compared to the province and the country as a whole. Air pollution has not been demonstrated to be causally associated with all of these cancer sites, so this general increase in cancers in the City of Saint John must have another explanation.

Health Status Report 2001 also contains health statistics related to other respiratory diseases, such as asthma, bronchitis, and emphysema. (These data do not include lung cancer.) The number of hospital discharges for respiratory diseases between April 1, 1999 and March 31, 2000 for New Brunswick and its seven Health Regions is provided. These data are reported as frequencies or counts, not rates of discharge. The percentage of a particular respiratory disease

as part of the total number of discharges due to respiratory disease is also reported. For New Brunswick as a whole, 14,390 discharges for respiratory diseases occurred, with 10% due to asthma, 17% due to bronchitis/emphysema, and 73% due to all other respiratory disease categories. For Health Region 2, 2,861 discharges occurred over the same period, with 7% due to asthma, 16% due to bronchitis/emphysema, and 77% due to all other respiratory disease categories. Thus, there appears to be a lower fraction of cases of asthma and bronchitis/emphysema (based on hospital discharge data) in Region 2, which contains Saint John City, compared to the province as a whole.

Average rates of hospital discharges per 1,000 population, for respiratory disease in general and chronic obstructive pulmonary disease (COPD) in particular, are also reported for New Brunswick and its seven Health Regions for the five-year period 1993/1994 – 1997/1998. In Region 2, the average number of hospital discharges per 1,000 population for all types of respiratory disease was lower than the provincial average, although not statistically significantly so. For the province as a whole, 22.4 and 20.2 discharges were reported per 1,000 males and females, respectively, for all types of respiratory diseases. For Region 2, 19.2 and 16.9 discharges were reported per 1,000 males and females, respectively. For discharges due solely to COPD, 6.8 and 6.3 were reported per 1,000 males and females, respectively, for the province as a whole. For Region 2, 5.6 and 4.8 COPD discharges were reported for males and females, respectively. Thus, COPD appears to be less prevalent in the region in which Saint John is situated compared to the Province as a whole.

Age-standardized mortality rates due to respiratory disease for Canada, New Brunswick, and the New Brunswick Health Regions are also reported in the *Health Status Report 2001*. These rates are for the year 1996, the most current data available at the time of the report. The rates of all respiratory disease deaths per 100,000 population for Canada, New Brunswick, and Health Region 2 are 59.8, 57.4, and 66.8, respectively. For deaths due solely to bronchitis, emphysema, and asthma, the rates are 6.1, 5.5, and 5.7 for Canada, New Brunswick, and Region 2, respectively. Therefore, respiratory disease mortality, in general, appears to be elevated in the Saint John Region compared to the province as a whole and to the country as a whole. However, the mortality rate due solely to bronchitis, emphysema, and asthma in the Saint John Region falls between that of the province and the country as a whole.

The New Brunswick Department of Health and Wellness collects data on various chronic health conditions, including asthma, to track the overall health status of the provincial population. In *Health Status Report 2001*, the Department's hospital discharge rates for 1997/1998 and 1999/2000 are provided, for all ages and for six 15-year age categories. These data are provided in Table 5-33. For both time periods and for all ages, the rate of asthma-related hospital discharges per 100,000 for New Brunswick as a whole are higher than for Region 2, which contains Saint John City. For instance, for all ages, the 1997/1998 rates are 234 for New Brunswick and 143 for Region 2. For 1999/2000, the hospital discharge rates are 192 for New Brunswick and 117 for Region 2.

According to Statistics Canada (1996-1997), the prevalence of asthma in New Brunswick is 6.6%, which is lower than the prevalence rates in all other provinces (7.1% to 8.5%) except for

Newfoundland, where its prevalence rate is 6.0%. According to Asthma Society of Canada (1999), 10-20% of children in Canada are reported to have asthma.

As reported in *Health Status Report 2001*, according to the New Brunswick Department of Health and Wellness 1997 Comprehensive Tobacco Reduction Strategy, tobacco use was the leading cause of preventable death in New Brunswick during that time period. Based on the Canadian Tobacco Use Monitoring Survey (CTUMS) for the year 1999, the rate of smokers (15 years old and higher) in New Brunswick (26%) was higher than the national average (25%) (Atlantic Health Sciences Corporation, 2001). The highest rate of smoking in the country was in Quebec (30%) and the lowest was in British Columbia (20%).

Other statistics confirm that smoking rates are elevated in Saint John City compared to the province and the country. According to New Brunswick Health and Wellness (2002), residents of Saint John have higher smoking rates than do residents of the province or the country as a whole. Smoking rates in Saint John among residents over the age of 15 were 29%. According to Statistics Canada (2001), the smoking rate in 1998-1999 in New Brunswick was 25% and in Canada was 23%. A survey performed in 1995 found that 63% of households had at least one regular smoker in them. Stieb et al. (2000b) also noted that there is "a generally higher prevalence of smoking in Saint John relative to elsewhere in Canada."

Because cigarette smoking causes lung cancer and chronic obstructive pulmonary disease (COPD) and environmental cigarette smoking can cause or exacerbate asthma, one must be careful in attributing cardiorespiratory disease in Saint John to air pollution. Clearly these diseases are multi-causal, and the high rates of smoking in Saint John must be taken into consideration when evaluating health status and the causes of adverse respiratory health effects.

In addition to *Health Status Report 2001*, Health Canada has produced a comprehensive report entitled *Statistical Report on the Health of Canadians* (Health Canada, 1999). The *Statistical Report* provides a description of health determinants and the health status of Canadians for a large number of health indicators. While it does not provide information specific to southwestern New Brunswick, it does provide data on Canada as a whole and on its provinces.

Table 5-34 is a compilation of various selected statistics presented in the *Statistical Report* for Canada and New Brunswick. These statistics range from hospital emergency department utilization rates to data on smoking patterns to mortality rates. A summary of how New Brunswick compares to Canada as a whole is given below:

- The utilization rate for emergency health services was reported to be lower in New Brunswick than in Canada as a whole. For 1995-96, the rate of hospital emergency department visits per 1,000 population for Canada was 433.1 compared to 190.9 for New Brunswick.
- For 1996, total health expenditures as dollars per capita was \$2,513 for Canada compared to \$2,371 for New Brunswick. Health expenditures include those in both the public and private sector (by the government as well as by individuals). As a percentage of gross domestic product, Canada spent 9.2% on health compared to 10.8% for New Brunswick

- Based on 1996-1997 data, the smoking experiences of Canada and New Brunswick appear to be similar. The percentages of current smokers, former smokers, and people who have never smoked are 28%, 29%, and 44% for Canada, respectively. For New Brunswick, 28%, 30%, and 42% of the population were categorized as current smokers, former smokers, and never smoked, respectively. The average number of cigarettes smoked daily in Canada over this time period was 17 compared to 18 for New Brunswick.
- Among the population of those 12 years and older, the prevalence of asthma in 1996-97 in Canada was 7% compared to 6% in New Brunswick. Asthma was defined as a chronic condition with a duration of at least six months.
- The incidence of new cancer cases and deaths for the year 1998 was also provided. The estimated rate of new cancer cases per 100,000 population was 501 males and 346 females for Canada as a whole. For New Brunswick, the rate of new cancer cases per 100,000 was 532 males and 335 females, respectively.
- The death rate from all cancers for Canada in 1998 was 232 males and 151 females per 100,000 population. For New Brunswick, the death rates were 255 males and 158 females per 100,000 population.
- The rate of hospital separations or discharges due to respiratory diseases was reported to be higher in New Brunswick than for Canada as a whole. For 1995-1996, the rate of hospital separations from respiratory diseases was 1060 per 100,000 for Canada compared to 2,000 per 100,000 for New Brunswick.
- For Canada for the year 1996, the age-standardized death rates for all respiratory diseases and for lung cancer were 58 and 49 deaths per 100,000, respectively. For New Brunswick, the same rates were 59 and 55 deaths per 100,000, respectively. The statistics for respiratory disease deaths presented here differ slightly from those presented by *Health Status Report 2001*. In that report, respiratory mortality rates for 1996 were 57 for New Brunswick and 60 for Canada. Both documents conclude that mortality from respiratory diseases is roughly equal for New Brunswick and Canada as a whole, and lung cancer mortality is elevated in the province compared to the country.
- The rates of potential years of life lost due to death from a particular cause were also provided. Potential years of life lost emphasize the loss of life at an early age; deaths prior to age 70 are considered "early" or potential years of life that have been lost. For Canada, deaths due to cancer were estimated to be responsible for 1,098 years of life lost per 100,000 population compared to 1,126 for New Brunswick. For potential years of life lost due to respiratory disease, the rate was 113 per 100,000 for Canada compared to 77 per 100,000 for New Brunswick.

In conclusion, various risk factors contribute to the incidence of the different types of cancer and respiratory diseases. These include age, lifestyles such as smoking and alcohol consumption, genetic predisposition, and environmental and occupational exposures. While unable to account for the role of such risk factors in the data summarized in this report, it appears that lung cancer incidence and mortality are higher in New Brunswick and Saint John City than in Canada as a whole. However, these same statistics are lower in the Saint John Area than in the province and in the country as a whole. A similar trend is seen for all cancers combined and many specific types of cancer. Because air pollution has not been shown to be associated with all of these many cancer sites, this general increase in cancers in the City of Saint John must have another

explanation. It also appears that the incidence of specific respiratory diseases such as asthma and COPD is lower in Health Region 2 (which contains Saint John Area and Saint John City) than in the province as a whole. This is based on hospital discharge data. While smoking rates for New Brunswick as a whole appear to be roughly the same or slightly higher than in Canada as a whole, the prevalence of smoking in Saint John is higher than in the province or the country as a whole. Data on specific respiratory disease rates in the sub-areas of Health Region 2 were not reported.

5.10 Cardiorespiratory Health Effects Epidemiology Studies

In this section, studies from Canada and the United States, which compare air pollution levels to adverse health outcomes, are discussed.

5.10.1 General Discussion of Statistical Studies Showing Associations Between Air Pollution and Adverse Health Outcomes

Many studies have been performed in Canada and the United States over the last ten years in an attempt to determine if air pollutants have caused various specific acute and chronic health effects in human populations. Air pollutants have generally been recognized to cause adverse health effects for years, and many regulatory agencies have been in place to protect human health. However, these recent studies have been performed to determine if various air pollutants are harmful at levels previously thought to be safe and if standards should be set at lower levels. These studies have focused on one or more of the following air pollutants: ozone, carbon monoxide, sulphur dioxide, nitrogen dioxide, sulfate, acidic particles, and respirable particulate matter ($PM_{2.5}$, PM_{10} , $PM_{10-2.5}$).

Because all of these pollutants are invariably present in the air at the same time and because the levels of many of them are highly correlated, the design and execution of such studies is very difficult. As such, these studies are statistical in nature. In such studies, researchers compare various indicators of health, such as health complaints, hospital admissions, emergency department admissions, or deaths on a day by day basis with the concentrations of one or more air pollutants to see if there is a statistical association between the health indicator and the pollutant concentration. If there is a statistically significant association, some researchers have presented this information as an indication that the air pollutant caused the adverse health outcome. For instance, if hospital admissions for asthma are higher on days when ozone levels are higher, the researchers may conclude that ozone *caused* the asthma hospital admission. Such associations do not actually prove a cause and effect relationship, but regulatory agencies are justified in assuming a causal relationship when the number of studies forms a sufficiently large data base.

The studies are, of course, very complex to perform. First, the researcher must make a decision as to what metric (measurement) of air pollution level is relevant to study. There are many choices, such as maximum daily concentration of the pollutant, average daily concentration, average concentration over two days, and so forth. In addition, researchers have found in earlier studies that health outcomes are not particularly correlated for many pollutants with the level in the

air on the day that the outcome occurred, such as the day that a person was admitted to a hospital emergency department. Instead, the concentration on the previous day or several days earlier is more strongly associated with the health outcome than is the current day's pollutant concentration. This is logical and biologically plausible, because a day of extremely poor air quality could trigger a health crisis in an individual that might result in their going to the hospital a day or two later. The difficulty in performing these statistical studies lies in defining the *lag time* that will be applied to the concentration data before determining if the pollutant concentrations are correlated with the health outcomes.

Many studies are performed with several different ways of summarizing the air pollutant data (daily maximum, daily average, 2-day daily average, 3-day daily average, etc.) and several different ways of lagging the data (0 days up to 10 days). For each pollutant studied, the approach that results in the highest statistical correlation is determined, and this approach is presented as the final result even if the approach is very different for each pollutant studied and each health outcome studied. Thus, the quantitative significance of these results is subject to some criticism. For instance, when one has a data set and performs endless different correlation analyses, sooner or later some of the correlations will register as *statistically* significant even though they may not be *biologically significant*. After all, a correlation is often defined as statistically significant if the probability is less than 1 in 20 that it occurred by chance alone. With this definition, the chances are high that some statistical tests will be statistically significant even if they are not biologically significant.

In fact, the authors of some of these studies admit this fact in a paper published in 1997 (Delfino, et al. 1997) when discussing a statistically significant association between same day PM_{10} and nonrespiratory hospitalizations in Montreal: "...given that 90 tests of relationships between nonrespiratory visits and air pollution were performed for the 1993 data set, one or two $p < 0.02$ associations are to be expected by chance alone."

Recent scientific research that has studied the relationship between air pollutant concentrations and adverse health effects in human populations are discussed below. In many cases, some of the shortcomings of the studies are described. Specifically, many of the results of the studies are not always replicated from one study to another. For instance, in one study ozone may be shown to be highly correlated with hospital admissions with respirable particulate matter not being associated, but in another study the opposite may be true. The shortcomings are not presented here to make the argument that air pollution has no effect on adverse health outcomes. On the contrary, it is assumed that air pollutants and health are causally linked. Instead, the shortcomings are presented to show that the study of the causes of health outcomes in populations is very difficult and that the quantitative results presented in each paper must be viewed as a general indication of a possible or likely causal link between air pollution and health and not as a solid quantitative predictor of the specific number of cases of one or another disease that can be expected to be seen if air pollution levels increase or decrease by a specific quantitative amount.

For instance, some of the papers described below present a number that defines a certain percent of the cases of a certain disease that were attributable to a certain level of one specific air

pollutant. However, these quantitative estimates vary widely from study to study, and they vary within a study when individual pollutants are statistically evaluated alone or when the full suite of air pollutants are simultaneously evaluated with complex statistical techniques. In some cases, a pollutant is shown in a single pollutant model to be associated with a specific health outcome, but when other pollutants are introduced into the analysis, the effect of the first pollutant is greatly reduced or disappears entirely. This phenomenon can occur, because the concentrations of different air pollutants are often highly correlated. So, if, for instance, ozone were to cause a specific health effect and particulate matter levels were high on days when ozone levels were also high, then an initial statistical analysis with particulate matter might indicate that particulate matter was associated with the health effect, when in fact, it was not. If a more complex analysis were performed with both ozone and particulate matter, it might be shown that the variability in ozone levels was associated with the variability in the health outcome and particulate levels did little if anything to add to the explanatory power of the statistical analysis. In such a case, one really would not be able to tell whether ozone or particulate matter or a combination of the two actually caused the health outcome that was studied.

5.10.2 Discussion of Recent Canadian Scientific Research Showing Associations Between Air Pollution and Adverse Health Outcomes

Of the Canadian studies, many have been performed in Saint John. These studies are discussed first, followed by a discussion of other Canadian studies.

5.10.2.1 Studies in Saint John, New Brunswick

New Brunswick Health and Wellness (2002). Local health authorities have summarized the recent scientific literature and have concluded that asthma symptoms can be aggravated by air pollution. With regard to chronic bronchitis and emphysema (called chronic obstructive pulmonary disease, or COPD) is caused mainly by smoking but can be worsened by several factors, including air pollution. The association between air pollution, if any, and heart diseases is "not fully understood."

Conclusion: Health authorities stated that asthma can be aggravated by air pollution, and the chronic obstructive pulmonary disease (COPD) is mainly caused by smoking, but can be aggravated by air pollution.

Brook et al. (1995). Daily (24 hour) measurements of fine particle acidity, sulfate, ammonium and total nitrate were collected at four locations in St. John, New Brunswick from June 1994 through October 1994. The locations were downtown St. John, Quispamis, Forest Hills, and at an upwind background location at Point Lepreau, 40 km southwest of the Forest Hills station. Daily PM₁₀ and PM_{2.5} measurements were taken at Quispamis, Forest Hills, and Point Lepreau.

The researchers found that the average PM₁₀ concentration was 15 ug/m³ at Forest Hills and 14 ug/m³ at Quispamis. Average PM_{2.5} concentration was 9 ug/m³ at both Forest Hills and Quispamis.

Acidity, sulfate, PM_{10} and $PM_{2.5}$ were higher in the eastern and northeast areas of the city compared to downtown St. John. The authors conclude: "Given that there are no significant SO_2 sources on the west side of the city, it is not unreasonable to assume that, on average, PM concentrations decrease to LP levels within west Saint John. Past PM_{10} measurements collected by the New Brunswick Department of Environment (NBDOE) tend to support this assumption. Mean annual average 24 hour concentrations were $19 \mu g m^{-3}$ at FH [Forest Hills] $22 \mu g m^{-3}$ at DT [downtown], $17 \mu g m^{-3}$ just west of downtown and $13 \mu g m^{-3}$ in extreme west Saint John. Thus, historically a relatively rapid decrease in PM_{10} across west Saint John was observed and concentrations in extreme west Saint John were similar to those we observed at LP." Note: LP was the designation for the background monitoring station at Port Lepreau.

Conclusion: The results of this study support the conclusion that the Coleson Cove Generating Station is not a major contributor to the air pollution in Saint John. Because pollutant levels increase from west Saint John to east and northeast Saint John, sources within the city are major sources of the measured air pollution in Saint John.

Stieb et al. (1995). In this study, some preliminary results from the Saint John Particle Health Effects Study are presented. Statistical analyses of potential associations between hospitalizations and measures of air pollution status were not presented. No conclusions were made regarding the associations, if any, between respirable particulate levels and hospitalizations. The design of the study was discussed as was initial descriptive data about emergency department visits based on surveys.

Conclusion: No specific relevant conclusions were presented in this study.

Stieb et al. (1996a). In this study, the relationship between personal measurements of sulfates and particle acidity and fixed site measurements was studied in Saint John. The objective was to determine if average personal exposure was accurately reflected by measurements at fixed site monitors, so that such fixed site measurements could be reliably used in epidemiology studies. In the study population of adults visiting the emergency department with cardiorespiratory disease, fixed site sulfate monitors appeared to accurately reflect daily variability in average personal sulfate exposures. Thus, fixed site monitors can be used in epidemiology studies to determine if there is a relationship between health outcomes and sulfate exposures. However, personal exposure to particle acidity did not appear to be well-represented by fixed site monitors.

Conclusion: No specific relevant conclusions were presented in this study.

Stieb et al. (1996b). The association between emergency department admissions of people complaining of asthma symptoms and ozone, sulphur dioxide, nitrogen dioxide, sulfate, and Total Suspended Particulate was evaluated in Saint John, New Brunswick during the months of May through September for the years 1984 through 1992. The mean daily one hour maximum ozone level was 42 ppb; and the mean daily sulfate concentration was $6 \mu g m^{-3}$. A statistically significant association was found between emergency department visits of people complaining of asthma symptoms and ozone levels two days prior, but no relationship was seen between ozone and any

other respiratory complaints, such as cough, congestion, wheeze, shortness of breath, or difficulty breathing.

Strong evidence of a threshold for ozone's effects was presented. According to the authors: "The effect of ozone appeared to be reduced or absent when ozone concentrations were below 75 ppb." No associations were found between asthma emergency department visits and sulphur dioxide, nitrogen dioxide, sulfate or Total Suspended Particulate.

A shortcoming of the study is that the positive association was seen between ozone concentrations and emergency visits for asthma "complaints" and not doctor-diagnosed asthma. No association was seen for other complaints that might be due to asthma, such as cough, congestion, wheeze, shortness of breath, or difficulty breathing. Furthermore, according to the authors: "...having a presenting complaint of asthma was a relatively insensitive indicator of the presence of asthma as assessed by a physician, accounting for less than half of visits with a discharge diagnosis of asthma. It is unclear to what extent and in what direction this might bias our estimate of the effect of ozone on asthma." Thus, the people who report at the emergency department that they are experiencing an asthma attack may represent a subset of asthmatics who are especially sensitive to ozone's effects.

Conclusion: Ozone at high levels was the strongest predictor of emergency department visits of people complaining of asthma symptoms in Saint John. No information was provided on the effects, if any, of particulate matter. Evidence of an effects threshold was demonstrated.

Stieb et al. (2000a). This study of cardiorespiratory episodes among patients visiting emergency departments in Saint John, New Brunswick between July 1994 and April 1996 found an "unexpectedly high proportion of ED [emergency department] visitors for respiratory conditions who were either daily smokers or who reported a regular smoker in the household, relative to results reported elsewhere for asthma.... This could be accounted for by a generally higher prevalence of smoking in Saint John relative to elsewhere in Canada." Also, they found that the most common primary trigger of respiratory disease episodes was viral upper respiratory tract infection. This fact was consistent with other published literature on asthma. Thus, smoking and exposure to environmental tobacco smoke and viral infections are important causative factors in triggering asthma symptoms that require hospitalization. Regarding air pollution, the authors state that the strongest association has been found in the literature between air pollution and hospital admissions with a lag of 0-2 days prior to the visit. This statement does not match the conclusions of other papers written by these authors where reported lag times varied widely from pollutant to pollutant and disease to disease.

Conclusion: Smoking and exposure to environmental tobacco smoke and viral infections were the main triggers of respiratory disease in Saint John.

Stieb, et al (2000b). The possible association between air pollution (ozone, sulphur dioxide, nitrogen dioxide, sulfate, carbon monoxide, PM₁₀, PM_{2.5}, fungal spores, pollen, and other air pollutants) and emergency department visits for numerous respiratory and cardiac conditions was investigated for the period 1992 through 1996 in Saint John, New Brunswick. In single pollutant

analyses, statistically significant positive associations were seen between respiratory emergency department visits and ozone, sulphur dioxide, PM_{10} , $PM_{2.5}$, total reduced sulphide and sulfate. Significant negative associations were seen with carbon monoxide, hydrogen sulphide, nitrogen dioxide, acidity, and coefficient of haze. For asthma, in particular, statistically significant positive associations were seen for ozone, sulphur dioxide, carbon monoxide, hydrogen sulphide, total reduced sulfide, PM_{10} , $PM_{2.5}$, acidity, and sulfate. Negative associations were seen with nitrogen dioxide and coefficient of haze. For chronic obstructive pulmonary disease (COPD), in particular, statistically significant positive associations were seen with carbon monoxide, hydrogen sulphide, nitrogen dioxide, ozone, sulphur dioxide, total reduced sulfide, PM_{10} , $PM_{2.5}$, and sulfate. Negative associations were seen with acidity and coefficient of haze. Similar effects were seen for cardiac emergency department visits. Ozone, hydrogen sulphide, sulphur dioxide, PM_{10} , and $PM_{2.5}$ were positively associated, and acidity, sulfate, and coefficient of haze were negatively associated.

Because individual air pollutant concentrations are correlated with each other, complex multi-pollutant analyses were performed to determine which air pollutants explained the most variation in the emergency department visits. In the final multi-pollutant models, ozone accounted for 4% of respiratory emergency department visits and sulphur dioxide for 4% of the visits. (Nitrogen dioxide was negatively associated with 4% of the visits). For cardiac emergency department visits, ozone accounted for 16% of visits and sulphur dioxide for 5%. In a multi-allergen model of warm season asthma visits, three specific types of fungal spores accounted for 13% of emergency department visits. The authors concluded that variability in ozone and sulphur dioxide concentrations consistently explained the variability in daily emergency department visits for both respiratory and cardiac effects, and the variation in respiratory particulate matter levels did little to improve the statistical analysis. Airborne fungal spores were significantly associated with warm season asthma visits.

A major shortcoming of this study is that the effects of air pollution on emergency department visits were analyzed with many different measurements of air pollution concentrations such as maximum daily value, average daily value, and average five day value, and many different lag times between the air pollution measurement and the daily emergency department admission rates, ranging from 0-10 days of lag. According to the authors, these "repeated hypothesis tests could result in spurious positive results at conventional levels of statistical significance." In fact, the most significant single pollutant results which are reported in the paper vary in lag time for 0-10 days among pollutants and even for a single pollutant when respiratory and cardiac emergency department admissions are compared. For instance, ozone gives the highest association with cardiac visits when lagged six days and the highest association with respiratory visits when lagged one day. However, if one looks at the association of ozone lagged six days with respiratory visits, the association is negative. That is, high ozone levels six days before is associated with fewer emergency department visits whereas high ozone levels 1-5 days before is associated with greater numbers of emergency department visits.

Conclusion: Ozone and sulphur dioxide were associated with emergency department admissions for respiratory and cardiac conditions in Saint John. In single pollutant analyses, positive associations were reported for some measures of respiratory particulate matter, but in the multi-pollutant models all the variability in emergency department admissions was explained by ozone

and sulphur dioxide. The paper provides little support for an association between respiratory particulate matter and acute hospitalizations for respiratory or cardiac symptoms. Fungal spores were shown to be significantly associated with warm season asthma emergency department admissions.

Summary of Studies of Health Outcomes in Saint John. Several epidemiology studies have been performed in Saint John to determine if the variability in adverse respiratory or cardiac health effects might be associated with variability in air pollutants. In one study (Stieb et al., 1996b), asthma emergency department admissions were found to be statistically significantly associated with ozone levels. No effects of particulate matter were shown. In another, (Stieb et al., 2000a), emergency department admissions for respiratory diseases was shown to be associated with smoking and viral upper respiratory tract infections. Lastly, Stieb et al. (2000b) studied emergency department admissions for respiratory and cardiac diseases and found that ozone and sulphur dioxide could explain the variability in admissions. No effects were seen for respiratory particulate matter in multipollutant analyses. Fungal spores were also associated with adverse health effects during warm months.

In conclusion, the studies performed in Saint John provide little support for the hypothesis that the variability in the rates of adverse health outcomes is attributable to the variability in the concentration of respiratory particulate matter, such as PM_{10} or $PM_{2.5}$. Studies performed elsewhere in Canada and the United States have shown that part of the variability in health outcome rates may be associated with varying rates of particulate matter concentrations, but the Saint John studies differ in their conclusions from these other studies. The conclusions of the Saint John studies point to ozone, sulphur dioxide, fungal spores, and smoking as potential causes of respiratory and cardiac disease.

5.10.2.2 Other Canadian Studies

Environmental Canada/Health Canada (2000). The Canadian Environmental Protection Act (CEPA) requires federal Ministers of the Environment and Health to publish a Priority Substances List that identifies substances that may be harmful to the environment or constitute a danger to human health. Substances on this list must then be evaluated to determine if they are "toxic" or are capable of becoming "toxic" in accordance with a specific definition of toxicity in Section 64 of the Act. If the Ministers identify a substance as "toxic", that substance may be regulated in accordance with the Act.

In the document entitled "Priority Substances List Assessment Report: Respirable Particulate Matter Less Than or Equal to 10 Microns", the Ministers conclude that respirable particulate matter (PM_{10} and $PM_{2.5}$) is considered to be "toxic" as defined in Section 64 of CEPA 1999. The document summarizes the scientific data and information considered by the Ministers in making this determination.

The document summarized 19 studies that investigated the relationship between short term variations in particulate matter levels with mortality. 16 studies were summarized that investigated the relationship between particulate matter levels and hospitalizations and emergency department

visits for cardio-respiratory disease. Other studies were summarized that addressed the association of short-term particulate matter concentrations on respiratory symptoms, such as lung function test performance. Several studies also addressed long term particulate matter concentrations and chronic health effects.

The Ministers evaluated the available data and concluded: "The database supports, therefore, a casual relationship between current ambient PM_{10} and $PM_{2.5}$ exposure and adverse health effects and provides a reasonable basis for preventive action." This conclusion is required for regulatory action, and it forms the basis for the Canada Wide $PM_{2.5}$ standard of $30 \mu g/m^3$ which was recently adopted.

The Environment Canada/Health Canada (2000) document concludes that the various published studies present an adequate weight of evidence to take regulatory action to reduce exposures to respirable particulate matter. However, the various studies are not without limitations and uncertainties.

For instance, Environment Canada/Health Canada (2000) reports that there is uncertainty in exposure assessment for particulate matter. Also, "there is concern for possible confounding from exposure to co-occurring (and often highly correlated) pollutants, in which case the increased risk could be ascribed to the wrong agent..." The document also notes that the strength of some of the associations is weak, and the mechanism of action of particulate matter on health effects has not been defined.

Despite the uncertainties, the Ministers have concluded that the studies provide strong enough evidence to define particulate matter as "toxic" and to issue a Canadian Wide Standard for $PM_{2.5}$.

Burnett et al. (1994). The authors investigated the potential effects of ozone and sulfate on acute respiratory health in Ontario from May to August over the period 1983 to 1988. Ambient concentrations of ozone from 22 monitoring stations and sulfate from 9 monitoring stations were compared to emergency room admissions at 168 local hospitals. Mean concentrations of ozone from May to August of 1983-1988 ranged from 32 ppb to 70 ppb (1 hour maximum values). Mean sulfate levels over the same period ranged from $3 \mu g/m^3$ to $12 \mu g/m^3$ (daily values). Statistically significant associations were found between hospital admissions for asthma, COPD, and infections and concentrations 1, 2 and 3 days prior to admission. Respirable particulate matter and other air pollutants were not evaluated.

Ozone was a stronger predictor of hospital admissions than sulfate with ozone explaining 5% and sulfate an additional 1% of admissions. Ozone and sulfate were associated with 7% of asthma admissions and 6% of COPD admissions. For infants, ozone and sulfate were associated with 13% of asthma and (-)16% of COPD admissions. The fact that the model predicted that increases in ozone were associated with *decreases* in COPD admissions for infants demonstrates the shortcomings of such epidemiology studies and argues that the results of such studies be used with care.

Another shortcoming of the study is that statistical associations were only observed during periods of very poor air quality. According to the authors, air pollution was not related to hospital admissions during the winter months of December to March when ozone and sulfate levels are low. A lack of demonstrated associations during periods of low air pollutant concentrations could be argued as evidence of an effects threshold, a level of ozone and sulfate below which acute respiratory effects do not occur.

Lastly, in this study, measurements of air quality were chosen to be the daily 1 hour maximum concentration of ozone and the daily average sulphate concentration. Other studies by the same authors have used different metrics, such as 1-, 2- or 3-day average pollutant concentrations. Concentrations were also lagged with respect to hospital admissions by 1, 2, or 3 days, depending on which lag period gave the highest statistical association.

Ozone at high concentrations was the strongest predictor of acute hospital admissions for asthma and COPD. No information was presented on the effects, if any, of respirable particulate matter. Evidence of an effects threshold was demonstrated. Highest statistical associations were found for 1 hour maximum ozone concentrations lagged 1 and 3 days and daily average sulphate concentrations lagged 1 day.

Burnette et al. (1999). The authors investigated the potential effects of ozone, sulphur dioxide, nitrogen dioxide, carbon dioxide, and respirable particulate matter (PM_{10} , $PM_{2.5}$, $PM_{10-2.5}$) on hospital admissions for respiratory, cardiac, cerebral vascular, and peripheral vascular diseases in metropolitan Toronto from 1980 to 1994. Air pollutant concentrations were obtained from four monitoring stations. The average daily particulate concentrations for the period were $30 \mu g/m^3$ for PM_{10} , $18 \mu g/m^3$ for $PM_{2.5}$ and $12 \mu g/m^3$ for $PM_{10-2.5}$.

In single pollutant analyses, all three measures of respirable particulate matter were associated with increases in respiratory and cardiac hospital admissions, but when the analysts controlled for gaseous pollutants, the increases in hospitalizations attributable to respirable particulate matter were reduced by a factor of four in magnitude, indicating that gaseous pollutants were more highly associated with hospital admissions than particulate matter. According to the authors, of the excess hospitalizations that were attributable to air pollution, 40% were attributable to nitrogen dioxide, 20% to ozone, 17% to carbon monoxide, 12% to $PM_{2.5}$, 8% to $PM_{10-2.5}$, and 3% to sulphur dioxide.

For excess asthma hospitalizations, 5% was attributable to ozone, 4% to carbon monoxide, and 4% to $PM_{10-2.5}$. For excess COPD hospitalizations, 6% was attributable to ozone, 4% to $PM_{10-2.5}$, and 3% to carbon monoxide.

A shortcoming of this study is that the authors considered multiple day averages of air pollutant concentrations varying from 1-3 days and concentrations lagged with respect to hospital admission by 0, 1 or 2 days. According to the authors: "We ran nine corresponding regression models for each pollutant on each disease, and we selected the lag and averaging period with the largest ratio of the log-relative risk to standard error (T value) for further analysis." Results vary widely for each pollutant and each disease.

For instance, for asthma, it was found that $PM_{2.5}$ and PM_{10} were most highly associated with increased hospitalizations when 3-day average concentrations were lagged 0 days, but $PM_{10-2.5}$ was most highly associated with increased hospitalizations when 3-day average concentrations were lagged 2 days. For COPD, it was found that $PM_{2.5}$ was most highly associated with increased hospitalizations when 2-day average concentrations were lagged 1 day; $PM_{10-2.5}$ was most highly associated with increased hospitalizations when 3-day average concentrations were lagged 2 days; and $PM_{10-2.5}$ was most highly correlated when 1-day average concentrations were lagged 2 days.

Total respiratory and cardiac hospitalizations were found to be strongly statistically associated with gaseous air pollutants (nitrogen dioxide, ozone, carbon monoxide, and sulphur dioxide) and less strongly associated with respirable particulate matter. Highest statistical associations for asthma were found for 3-day average ozone concentration lagged 1 day, 3-day average sulphur dioxide concentration lagged 2 days, and 3-day average $PM_{10-2.5}$ concentration lagged 0 days.

Burnett et al. (1998a). The authors reported statistically significant associations between daily fluctuations in nonaccidental mortality (cardiac and other nonaccidental were evaluated separately) and ambient levels of CO, NO₂, SO₂, coefficient of haze, TSP, sulfates, and *estimated* $PM_{2.5}$ and PM_{10} in Toronto during the period 1980-1994. Increases in relative risk of death were associated with all of the parameters measured when analyzed separately, because different measures of air pollution were all correlated with each other. In multipollutant analyses, only carbon monoxide and TSP gave statistically significant associations. As noted by the authors: "...the effects of this complex mixture of air pollutants could be explained by the levels of carbon monoxide (CO) and total suspended particulates." Of the daily nonaccidental deaths, the authors attributed five times more to carbon monoxide than to TSP.

As with other studies by these researchers, different metrics for each air pollutant and different lag times were all analyzed to identify the measurement type and the lag period that would give the strongest association. The best fit was found with ozone for a 1-day average and a 1 day lag. For sulphur dioxide, sulphate, PM_{10} and TSP, the best fit was found with a 1-day average and a 0 day lag. For carbon monoxide and $PM_{2.5}$, the best fit was found with a 2-day average and a 0 day lag. As noted elsewhere in this report, these metrics and lag times do not agree with those identified in other studies by the same authors.

Burnett et al. (1995). In this study, the authors studied the fluctuations in sulphate and ozone levels and hospital admissions for cardiac and respiratory diseases for Ontario for the years 1983-1988. Concentration metrics were maximum 1-hour ozone concentrations and daily average sulphate concentrations. Four different lag times were investigated. Sulphate concentrations lagged 3 days were not associated with cardiorespiratory admissions, but concentrations lagged 0, 1 and 2 days gave statistically significant associations with hospital admissions. For both respiratory and cardiac admissions, only sulphates recorded 1 day previous to admission were included in the final regression model. A positive association was found for all cardiac admissions, all respiratory admissions, asthma admissions, COPD, admissions, and respiratory infection admissions. No association was seen with ozone.

Dales et al. (2000). These authors studied the association between emergency department admissions for asthma in a children's hospital in Ottawa, Ontario from 1993-1997 and concentrations of pollen, fungal spores, ozone, nitrogen dioxide, sulphur dioxide, and sulphates. Hospital admissions were associated with ozone levels and fungal spore concentrations. An increase in hospital admissions of 9% was associated with fungal spores. Thus, the authors concluded: "...fungal spores account for a significant proportion of the asthma exacerbations in children that prompt an emergency department visit."

Burnett et al. (2000). In this paper, the authors admit that there is a serious issue of confounding in performing population studies that attempt to find associations between adverse health effects and air pollution levels. Although "it is not possible to completely eliminate" the concern about confounders and covarying pollutants, the authors conclude from all of the population studies that "confounding cannot explain all the observed impacts." The authors further state that regulatory actions that were taken in recent years were done as a matter of prudent public policy in the face of remaining uncertainties. Specifically, "it was judged by several government bodies that it would be prudent to act on the assumption that the association between particulate matter and cardiorespiratory disease is causal."

Burnett et al. (1998b). In this study, the authors studied the fluctuations in levels of carbon monoxide, sulphur dioxide, nitrogen dioxide, ozone, and $PM_{2.5}$ and nonaccidental mortality in eleven Canadian cities for the years 1980-1991. As with other studies performed by these authors: "the relative risk of death attributable to each pollutant and city separately were determined using generalized additive models for longitudinal count data for single, two-day, and three-day averages of the pollutant concentration lagged zero, one, and two days. A single averaging time and days lagged was selected for each pollutant and city based on the largest relative risk...."

The results of the study were that nitrogen dioxide was associated with a 4.1% increased risk of mortality, with ozone, sulphur dioxide and carbon monoxide explaining 1.8%, 1.4%, and 0.9% in multiple pollutant regression models averaged over the eleven cities. According to the authors, "there is no apparent association, however, between total risk of the air pollution mix, based on multiple pollutant models and $PM_{2.5}$ concentrations (plot not shown), suggesting that the four gaseous pollutants can adequately explain daily variations in mortality rates and that the addition of $PM_{2.5}$ is unlikely to add any predictive power." This result does not agree with Burnett et al. (1998a) in which carbon monoxide alone explained 4.7% of increased risk of mortality, and particulates were associated with an additional increase in risk but other gaseous pollutants were not associated with increased risk.

As noted elsewhere the fact that the authors evaluated many different ways to measure pollutant concentrations and many different lag times and did so independently for each city is a shortcoming of the study. Thus, the statistically significant associations between pollutants and mortality are reported even though the biological plausibility is not clear. For instance, the associations reported for carbon monoxide and sulphur dioxide vary from 1-, 2- and 3-day average concentrations and from 0, 1, and 2 days of lag. It is perhaps understandable that one

pollutant might be direct acting within a day of an episode of high concentration and another pollutant might cause death after several days, but it is not understandable why the mechanism of action of a pollutant would vary so widely among eleven cities. As can be seen in Table 5-35, the authors would have us believe that each of these pollutants has a different mechanism of action depending on which city they are considering. In reality, the mechanism of action of pollutant concentrations on health outcomes should not be different for different cities. The quantitative results presented in this paper are an artificial result of the statistical manipulations used by the authors.

Delfino et al. (1997). In this study, the authors studied the fluctuations in levels of ozone, PM_{10} , $PM_{2.5}$ and sulphate and emergency room admissions for respiratory illnesses in Montreal for the summer months (June-September) for the years 1992 and 1993. Concentration metrics were maximum 1-hour and 8-hour ozone concentrations and daily average sulphate, PM_{10} , $PM_{2.5}$ and acidity concentrations. Three different lag times were investigated: 0, 1 and 2 days. No significant associations were found between pollutant concentrations and respiratory hospitalizations for 1992. For 1993, there were no significant associations between air pollution and respiratory hospitalizations for people between the age of 2 and 64. For children under the age of 2, only acidity was statistically significantly associated with hospitalizations. For people over 64 years of age, significant associations were separately seen with ozone (1 and 8 hour maximum values, lagged 1 day); PM_{10} (average daily concentration, lagged 1 day), $PM_{2.5}$ (average daily concentration, lagged 1 day), and sulfate (average daily concentration, lagged 1 day). When multipollutant models were evaluated, the association between particulate concentrations and hospitalizations was greatly reduced and the association between ozone and hospitalizations was only slightly reduced.

Burnett et al. (2001). In this study, the authors studied the fluctuations in levels of ozone, $PM_{10-2.5}$, $PM_{2.5}$, nitrogen dioxide, sulphur dioxide, and carbon monoxide and hospitalizations for respiratory illnesses for children under the age of 2 in Toronto for different seasons for the years 1980-1994. Concentration metrics were 1-, 2-, 3-, 4- and 5- day averaged maximum 1-hour ozone concentrations and 1-, 2-, 3-, 4- and 5- day averaged daily $PM_{10-2.5}$, $PM_{2.5}$, nitrogen dioxide, sulphur dioxide, and carbon monoxide concentrations. Six different lag times were investigated: 0, 1, 2, 3, 4, and 5 days.

Statistically significant associations were found between ozone, $PM_{2.5}$, $PM_{10-2.5}$, nitrogen dioxide, sulphur dioxide, and carbon monoxide in single pollutant and multiple pollutant models with differing averaging times and lag times. In the multiple pollutant model, ozone showed the strongest association with carbon monoxide next strongest.

Burnett et al. (2000). In this study, the authors studied the fluctuations in levels of ozone, PM_{10} , $PM_{10-2.5}$, $PM_{2.5}$, nitrogen dioxide, sulphur dioxide, carbon monoxide and sulphate and daily mortality in eight Canadian cities for the years 1986- 1996. Concentration metrics were maximum 1-hour ozone concentrations and 1-day averaged daily PM_{10} , $PM_{10-2.5}$, $PM_{2.5}$, nitrogen dioxide, sulphur dioxide, carbon monoxide, and sulphate concentrations. Six different lag times were investigated: 0, 1, 2, 3, 4, and 5 days.

In single pollutant models all air pollutants were statistically significantly associated with variation in mortality rates. Nitrogen dioxide, ozone, and carbon monoxide gave the highest associations. With multiple pollutant models, the associations of all individual air pollutants were reduced in magnitude, with ozone, nitrogen dioxide and $PM_{2.5}$ giving the highest associations.

Cakmak et al. (1998). In this study, the authors studied the fluctuations in levels of ozone and hospitalizations for respiratory disease in Toronto for the years 1981- 1991. The concentration metric used was the maximum daily 1-hour ozone concentrations. Six different lag times were investigated: 0, 1, 2, 3, 4, and 5 days. It was found that the ozone concentrations recorded two days prior to hospital admission were most strongly associated with respiratory hospitalizations.

Burnett et al. (1997a). In this study, the authors studied the fluctuations in levels of ozone, total particulate, coarse particulate, fine particulate, nitrogen dioxide, sulphur dioxide, carbon monoxide, acidity, and sulphate and daily hospital admissions in Toronto for the summer seasons of years 1992-1994. Concentration metrics were maximum 1-hour, daily 24- hour average, and 8- hour daytime average concentrations averaged over 1-, 2-, 3-, 4- and 5- day periods for ozone, nitrogen dioxide, sulphur dioxide, and carbon monoxide and 24 hour average daily concentrations averaged over 1-, 2-, 3-, 4- and 5- day periods for total particulate, coarse particulate, fine particulate, sulphate and acidity. Six different lag times were investigated: 0, 1, 2, 3, 4, and 5 days. For each air pollutant the authors evaluated up to 54 different statistical evaluations to identify the one that maximized the association between variability in pollutant concentration and variability in daily cardiorespiratory hospitalizations. This single worst case statistic was "selected for further analysis."

Single pollutant analyses showed statistically significant associations between all air pollutants and both cardiac and respiratory hospitalizations. "However, the association between the health outcomes and carbon monoxide, fine and coarse mass, sulfate levels, and aerosol acidity could be explained by adjustment for exposure to gaseous pollutants." The inclusion of any one of the particulate air pollutants in multiple regression models did not increase the magnitude of the associations. The authors concluded that ozone, nitrogen dioxide, and sulphur dioxide explained all of the air pollution related increases in hospitalizations. "Based on the results from this study, we cannot attribute any adverse health effects to particle mass or chemistry alone." Ozone was the single most important parameter that affected adverse health outcomes.

Burnett et al. (1997b). In this study, the authors studied the fluctuations in levels of ozone, nitrogen dioxide, sulphur dioxide, carbon monoxide, and coefficient of haze and daily hospital admissions for congestive heart failure in elderly people in ten Canadian cities for the years 1981- 1991. Concentration metrics were maximum 1-hour and daily 24- hour average concentrations for ozone, nitrogen dioxide, sulphur dioxide, coefficient of haze and maximum 1-hour and daily maximum 8-hour running average for carbon monoxide. Four different lag times were investigated: 0, 1, 2, and 3 days.

In single pollutant analyses, statistically significant associations were reported for carbon monoxide, nitrogen dioxide, sulphur dioxide and coefficient of haze. In multiple pollutant analyses, carbon monoxide and nitrogen dioxide still showed statistically significant associations,

but sulphur dioxide, ozone, and coefficient of haze were not associated with increased hospitalizations.

Brook et al. (1997). In this paper, concentrations of total suspended particulate, PM_{10} , $PM_{2.5}$, sulphate and inorganic constituents are summarized for numerous locations within the National Air Pollution Surveillance network for the period 1984-1993. Data from the Saint John station at Forest Hill is summarized. Over the period of study, the average $PM_{2.5}$ concentration in Saint John was $10.1 \mu g/m^3$ compared to the average level of $13.8 \mu g/m^3$ for all 19 stations throughout Canada. The average $PM_{10-2.5}$ concentration in Saint John was $7.3 \mu g/m^3$ compared to the average level of $12.1 \mu g/m^3$ for all 19 stations throughout Canada.

Metal data was not available for Saint John, but the median levels of metals nationwide were as follows:

Arsenic	$<0.001 \mu g/m^3$
Beryllium	No Data
Cadmium	<0.002
Chromium	0.002
Lead	0.052
Mercury	0.001
Nickel	0.002
Selenium	<0.0008
Vanadium	0.005

Burnett et al. (1997c). In this study, the authors studied the fluctuations in levels of ozone, nitrogen dioxide, sulphur dioxide, carbon monoxide, and coefficient of haze and daily hospital admissions for respiratory diseases in sixteen Canadian cities (including Saint John) for the years 1981-1991. Concentration metrics were maximum 1-hour, daily 24-hour average, maximum 8-hour running average, and 8-hour daytime average concentrations for ozone and maximum 1-hour concentration for nitrogen dioxide, sulphur dioxide, coefficient of haze, and carbon monoxide. Three different lag times were investigated: 0, 1, and 2 days.

In single pollutant statistical analyses, ozone, carbon monoxide, coefficient of haze, nitrogen dioxide, and sulphur dioxide were associated with increased respiratory hospitalizations. In multiple pollutant analyses, only ozone and carbon monoxide were associated with hospitalizations.

Watson (1997). In this paper, a critical review of the scientific data on ambient particles and health, which was commissioned by the Air and Waste Management Association, is discussed and the comments of a panel of experts regarding the critical review are presented. The critical review and the comments demonstrate that there are uncertainties remaining regarding the specific health effects caused by respirable particulate matter in the air.

5.10.2.3 Summary of Canadian Research Regarding Air Pollution and Adverse health Outcomes

The following section summarizes the results of the Canadian research studies discussed in Sections 5.9.2.2 and 5.9.2.3 above.

Hospitalizations

1. Burnett et al. (1994)

Effect Studied: Asthma hospitalizations

Major Association: ozone > sulphate

Metrics and Lag Times:

Ozone: 1 hour maximum; 1 + 3 day lag

Sulphate: Daily average; 1 day lag

2. Burnett et al. (1995)

Effect Studied: Hospitalizations for Cardiac and Respiratory Diseases

Major Association: sulphate (ozone not associated in multiple regression model)

Metrics and Lag Times:

Sulphate: 1-day average; 1 day lag

3. Stieb et al 1996b

Effect Studied: Asthma hospitalizations

Major Association: ozone (Sulphur dioxide, nitrogen dioxide, sulphate, TSP not associated with health effects in multipollutant model.)

Metrics and Lag Times:

Ozone: Maximum 1-hour; 2 day lag

4. Delfino et al. (1997)

Effect Studied: Respiratory Hospitalizations

Major Associations: $PM_{2.5} > PM_{10} > \text{ozone} > \text{sulphate}$ in single pollutant models, however, ozone was more strongly associated in multipollutant models;

Metrics and Time Lags:

PM_{10} : 1-day average, 1 day lag

$PM_{2.5}$: 1-day average, 1 day lag

Sulphate: 1-day average, 1 day lag

Ozone: 1-hour and 8-hour maximum, 1 day lag

5. Burnett et al. (1997a)

Effect Studied: Cardiorespiratory Hospitalizations

Major Associations: Ozone > nitrogen dioxide, sulphur dioxide (no association seen for total particulate, coarse particulate, fine particulate, carbon monoxide, sulphate, and acidity in multipollutant analyses)

Metrics and Time Lags:

Ozone: 3-day 8-hour daily average, 1 day lag for respiratory; 3-day 8-hour daily average, 2 day lag for cardiac;
Nitrogen dioxide: 5-day 8-hour daily average, 0 day lag for respiratory; 4-day 8-hour daily average, 0 day lag for cardiac;
Sulphur dioxide: 4-day average of 1-hour maxima, 0 day lag for respiratory; 4-day average of 1-hour maxima, 0 day lag for cardiac;
Carbon monoxide: 3-day average of 1-hour maxima, 2 day lag for respiratory; 2-day average of 8-hour averages, 0 day lag for cardiac;
Acidity: 2-day average of 24-hour averages, 0 day lag for respiratory; 4-day average of 24-hour averages, 2 day lag for cardiac;
Sulphate: 4-day average of 24-hour averages, 1 day lag for respiratory; 4-day average of 24-hour averages, 2 day lag for cardiac
Fine Particulate: 4-day average of 24-hour averages, 1 day lag for respiratory; 4-day average of 24-hour averages, 2 day lag for cardiac
Coarse Particulate: 5-day average of 24-hour averages, 0 day lag for respiratory; 4-day average of 24-hour averages, 0 day lag for cardiac

6. Burnett et al. (1997b)

Effect Studied: Cardiac Hospitalizations

Major Association: Carbon monoxide > nitrogen dioxide (ozone, sulphur dioxide, coefficient of haze not associated in multipollutant analyses)

Metrics and Time Lags:

Carbon monoxide: 1-hour maxima, 0 day lag;
Nitrogen dioxide: 24-hour average, 0 day lag
Ozone: 24-hour average, 2 day lag
Sulphur Dioxide: 24-hour average, 0 day lag
Coefficient of Haze: 1-hour maxima, 0 day lag

7. Burnett et al. (1997c)

Effect Studied: Respiratory Hospitalizations

Major Association: Ozone > carbon monoxide (nitrogen dioxide, sulphur dioxide, and coefficient of haze not associated in multipollutant analyses)

Metrics and Time Lags:

Ozone: 1-hour maxima, 1 day lag
Carbon monoxide: 1-hour maxima, 0 day lag;
Nitrogen dioxide: 1-hour maxima, 0 day lag
Sulphur Dioxide: 1-hour maxima, 0 day lag
Coefficient of Haze: 1-hour maxima, 0 day lag;

8. Burnette et al. (1999)

Effect Studied: Asthma hospitalizations

Major Association: ozone > PM₁₀, PM_{2.5} = carbon monoxide (sulphur dioxide, nitrogen dioxide not associated with health effects in multipollutant model)

Metrics and Lag Times:

Ozone: 3-day average; 1 day lag

Sulphur dioxide: 3-day average; 2 day lag;
PM_{10-2.5}: 3-day average; 2 day lag
PM_{2.5}: 3-day average; 0 day lag

Effect Studied: COPD hospitalizations

Major Association: ozone > PM₁₀, PM_{2.5} > carbon monoxide (sulphur dioxide, nitrogen dioxide not associated with health effects in multipollutant model)

Metrics and Lag Times:

Ozone: 3-day average; 2 day lag
Sulphur dioxide: 1-day average; 2 day lag;
PM_{10-2.5}: 3-day average; 2 day lag
PM_{2.5}: 2-day average; 1 day lag

9. Stieb, et al (2000b)

Effect Studied: Respiratory hospitalizations

Major Association: ozone > sulphur dioxide

Metrics and Lag Times:

Ozone: 5-day average; 1 day lag
Sulphur dioxide: 1-day average, 5 day lag

Effect Studied: Cardiac hospitalizations

Major Association: ozone > sulphur dioxide

Metrics and Lag Times:

Ozone: 1-day average; 6 day lag
Sulphur dioxide: 1-day average, 8 day lag

10. Burnett et al. (2001)

Effect Studied: Respiratory Hospitalizations

Major Associations: ozone > carbon monoxide > nitrogen dioxide > PM_{10-2.5} > PM_{2.5} > sulphur dioxide

Metrics and Time Lags:

Ozone: 5-day average of 1 hour maxima, 0 day lag
Carbon monoxide: 2-day daily average, 1 day lag
Nitrogen dioxide: 2-day daily average, 2 day lag
PM_{10-2.5}: 4-day daily average, 1 day lag
PM_{2.5}: 4-day daily average, 0 day lag
Sulphur dioxide: 1-day daily average, 3 day lag

Mortality Studies

1. Burnett et al. (1998a)

Effect Studied: Mortality

Major Association: carbon monoxide > TSP

Ozone: 1-day average; 1 day lag;
Carbon Monoxide: 2-day average, 0 day lag;

Sulphur dioxide: 1-day average, 0 day lag;
TSP, PM₁₀: 1-day average; 0 day lag
PM_{2.5}: 2-day average, 0 day lag
Sulphate: 1-day average, 0 day lag

2. Burnett et al. (1998b)

Effect Studied: Mortality

Major Associations: Nitrogen dioxide > ozone > sulphur dioxide > carbon monoxide
(PM_{2.5} not associated in multiple regression model)

Metrics and Lag Times:

Carbon monoxide: 1-, 2- or 3-day average; 0, 1, or 2 day lag (depending on city)

Nitrogen dioxide: 1-, 2- or 3-day average; 0, 1, or 2 day lag (depending on city)

Sulphur dioxide: 1-, 2- or 3-day average; 0, 1, or 2 day lag (depending on city)

Ozone: 1- or 2- day average; 0, 1, or 2 day lag (depending on city)

3. Cakmak et al. (1998)

Effect Studied: Mortality

Major Associations: ozone

Metrics and Time Lags:

Ozone: 1 hour maximum, 2 day lag

4. Burnett et al. (2000)

Effect Studied: Mortality

Major Associations: ozone > nitrogen dioxide > PM_{2.5} > carbon monoxide = sulphur dioxide > PM_{10-2.5}

Metrics and Time Lags:

Ozone: 1 hour maximum, 0 day lag

Carbon monoxide: 1-day daily average, 1 day lag

Nitrogen dioxide: 1-day daily average, 1 day lag

PM_{10-2.5}: 1-day daily average, 1 day lag

PM_{2.5}: 1-day daily average, 1 day lag

Sulphur dioxide: 1-day daily average, 1 day lag

Sulphate: 1-day daily average, 1 day lag

5.10.2.4 Conclusions of Canadian Research Regarding Air Pollution and Adverse Health Outcomes

As an example of the complexity of the results of air pollution human epidemiology studies, the concentration measurements and the lag times presented in the studies performed on Canadian cities by Canadian researchers are summarized for ozone in Table 5-36. In each study several ways of measuring the air pollutant concentration and several time lags were separately analyzed for one or more health outcomes. Out of 16 associations reported for ozone, there were 15 different concentration measurements and lag times. The conclusions of these studies can be used to argue that ozone is associated with adverse health outcomes, but one cannot use the quantitative results from the studies to define a dose-response factor that relates a specific ozone

exposure to a specific adverse health outcome. Because of all of the differences between one study and another, it is simply impossible to define the appropriate dose measurement and the appropriate lag time.

5.10.3 Discussion of Recent Non-Canadian Scientific Research Showing Associations Between Air Pollution and Adverse Health Outcomes

Several key studies for the United States are discussed below.

EPA (2001b)

In "Air Quality Criteria for Particulate Matter," the United States Environmental Protection Agency summarized recent scientific literature that was considered in the setting of the new National Ambient Air Quality Standards for $PM_{2.5}$ ($65 \mu g/m^3$, 24-hour; $15 \mu g/m^3$, annual average). Studies from around the world are summarized, including the Canadian studies discussed above. EPA's conclusions include the following. The evidence indicates that PM_{10} or one or more PM_{10} components is a "probably contributory cause of human health effects." There is "meaningful heterogeneity in the city-specific excess risk estimates for the relationships between short-term ambient PM_{10} concentrations and acute health effects." The reasons for the city-to-city variability are not understood at this time. Based on a smaller data base, EPA has concluded that $PM_{2.5}$ or one or more $PM_{2.5}$ components is a "probable contributing cause of observed PM-associated health effects." Coarse particles ($PM_{10-2.5}$) "may be a contributory cause of health effects in some locations." Concerning chronic exposures to particulate matter, EPA concluded: "notable uncertainties remain regarding the meaning, magnitude, and mechanisms for more chronic health effects of long-term PM exposures."

On the one hand, EPA has concluded that the effect size estimates for ambient particulate matter health effects are similar in many of the published studies, lending strength to the scientific weight of evidence that the associations between particulate matter concentrations and adverse health effects is causal. On the other hand, however, EPA notes a major shortcoming of the population studies summarized in the document:

One major methodological issue affecting epidemiology studies of both short-term and long-term PM exposure effects is that ambient PM of varying size ranges is typically found in association with other air pollutants, including gaseous criteria pollutants (e.g. O_3 , NO_2 , SO_2 , CO), air toxics, and/or bioaerosols. Available statistical methods for assessing potential confounding arising from these associations may not yet be fully adequate. The inclusion of multiple pollutants often produces statistically unstable estimates. Omission of other pollutants may incorrectly attribute their independent effects to PM. Much progress in sorting out relative contributions of ambient PM components versus other copollutants is nevertheless being made, and, overall, tends to substantiate that observed PM effects are at least partly due to ambient PM acting alone or in the presence of other covarying gaseous pollutants.

Regarding the chemical nature of particulate matter, EPA has concluded that not all PM is the same. "It is likely that differences in observed health effects will be found to depend as much on site-specific differences in chemical and physical composition characteristics of ambient particles as on differences in PM mass concentration." Because of this fact, EPA cautions: "The above reasons suggest that it is inadvisable to pool epidemiology studies at different locations, different time periods, with different population sub-groups, or different health endpoints, without assessing the consequences of these differences."

EPA also concluded that "certain classes of ambient particles may be distinctly less toxic than others and may not exert human health effects at typical ambient exposure concentrations or only under special circumstances. For example, particles of crustal origin may be relatively non-toxic under most circumstances compared to those of combustion origin."

In this summary document, EPA lists relative risk results from many different published studies. It should be noted that these are estimates of the association between particulate matter concentrations and adverse health outcomes from single pollutant studies. This is a major flaw in EPA's document. As noted above in the detailed summaries of many Canadian epidemiology studies, there are many studies in which authors presented associations between adverse health outcomes and measures of particulate matter that were initially seen in single pollutant analyses but which significant decreased or disappeared completely when other pollutants such as ozone or nitrogen dioxide were analyzed simultaneously. These initial single pollutant results are all presented by EPA to show that there is a large body of scientific evidence that justifies strict regulation of ambient particulate matter concentrations. However, EPA's document would have been more even-handed if it had listed the associations between adverse health outcomes and particulate matter after controlling for the effects of other pollutants.

For instance, the EPA document summarizes Burnett et al. (2000) and gives result for $PM_{2.5}$ showing a fraction of the variability in the deaths in eight Canadian cities that could be attributed to variability in the $PM_{2.5}$ concentration. What they fail to state is that Burnett et al. (2000) actually showed that associations between air pollution and mortality were 2.1 times greater for ozone, 2.4 times greater for nitrogen dioxide, and 1.3 times greater for carbon monoxide in single pollutant analyses. When multiple pollutant analyses were carried out, the association for $PM_{2.5}$ dropped by a factor of 1.6 compared to the result presented in EPA's table. In fact, of the association between air pollution and mortality from the multi-pollutant analysis, only 18% was due to $PM_{2.5}$. 28% was due to ozone, 19% was due to nitrogen dioxide, 12% was due each to sulphur dioxide and carbon monoxide, and 11% was due to $PM_{10-2.5}$.

Because they do not take into account the effects of other air pollutants, all of the relative risk estimates presented in EPA (2001b) should be viewed qualitatively as evidence of a link between airborne particulate matter and adverse health outcomes. They are not reliable quantitative estimates that explain the exact impacts of specific concentrations of particulate matter on health.

Pope et al. (2002)

Pope and co-workers studied the associations between day-to-day air pollution and increased risk of various adverse health outcomes in 500,000 adults throughout the United States in a study sponsored by the American Cancer Society. Air pollution data considered included $PM_{2.5}$, PM_{10} , PM_{15} , $PM_{15-2.5}$, total suspended particulates, sulfate, sulfur dioxide, nitrogen dioxide, carbon monoxide, and ozone.

In this study only single pollutant analyses were performed. That is, each measure of long-term air pollution was separately evaluated to determine its statistical association with all-cause mortality, cardiopulmonary mortality, lung cancer mortality, and all other cause mortality. All-cause mortality was statistically associated with long-term exposure to $PM_{2.5}$, sulphate, and sulphur dioxide. Cardiopulmonary mortality was statistically associated with long-term exposure to $PM_{2.5}$, PM_{10} , PM_{15} , sulphate, and ozone. Lung cancer mortality was statistically associated with long-term exposure to $PM_{2.5}$, sulphate, and sulphur dioxide. All other cause mortality was statistically associated with long-term exposure to sulphate and sulphur dioxide.

The conclusions of the paper are stated as follows: "Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality." Then, the results for $PM_{2.5}$ are specifically presented. However, it is clear from Figure 5 in the report that mortality is equally statistically associated with $PM_{2.5}$, sulphate, and sulphur dioxide and to a lesser degree with ozone, depending on the cause of mortality. If these pollutants are correlated, which has been demonstrated in many other scientific papers, then one cannot conclude whether the causative agent in adverse health outcomes is $PM_{2.5}$, sulphate, sulphur dioxide, or a combination of all three. No multipollutant models were presented in the paper that would attempt to determine which pollutant had the highest explanatory power to explain the variability in mortality rates.

The authors do, however, state: "Interestingly, measures of $PM_{2.5}$ were associated with all-cause [,] cardiopulmonary, and lung cancer mortality, but not with a" other mortality. However, sulfur oxide pollution (as measured by sulfate particles and/or sulfur dioxide) was significantly associated with mortality from all other causes in addition to all-cause, cardiopulmonary, and lung cancer mortality." Thus, the authors acknowledge that $PM_{2.5}$ is not the only air pollutant that yields statistical associations with mortality rates.

Regarding the magnitude of the increased risk reported for fine particle air pollution, the authors state that the risk is "obviously much smaller than the risk of cigarette smoking." In addition, the authors conclude that "mortality risks associated with fine particulate air pollution at levels found in more polluted US metropolitan areas are less than those associated with substantial obesity (grade 3 overweight), but comparable with the estimated effect of being moderately overweight (grade 1 to 2)."

In conclusion, this recent study finds that "long-term exposure to fine particulate air pollution common to many metropolitan areas is an important risk factor in cardiopulmonary mortality." However, the specific entity in the air that is a causal factor has not been identified, and no

quantitative estimation of mortality rates associated with increases or decreases in air pollution levels can be calculated from data in this paper.

Samet et al. (2000a)

Samet and co-workers reported on the results of the National Morbidity, Mortality, and Air Pollution Study in the United States. The study was designed to address the uncertainties surrounding the reported associations between particulate air pollution and daily mortality. These uncertainties included a lack of adequate treatment of confounding or effect modification by non-particulate pollutants that often correlate with particulate levels. Other uncertainties included the potential effects caused by the use of differing statistical methods by different researchers. Errors in air pollutant measurements including instrumental error and a lack of correlation between fixed monitor measurements and personal exposures were also considered as criticisms of the existing epidemiology studies. In addition, the issue of mortality displacement was discussed. This issue concerns the magnitude of the reduction in life expectancy. The study develops new methods that consider and examine the various criticisms discussed above and advances the science of air pollution epidemiology.

Samet et al. (2000b)

The National Morbidity, Mortality, and Air Pollution Study in the United States was a study of the potential association between PM₁₀, ozone, carbon monoxide, nitrogen dioxide, and sulphur dioxide and daily mortality in 20 and 90 of the country's largest cities. With regard to pollution concentration metrics and time lags, this study uses daily average pollutant concentrations and 1-hour maximum concentrations for ozone and lags the pollutant concentrations by 0, 1 and 2. Statistically significant associations were found between PM₁₀ when lagged 1 day and mortality rates. The effect was lessened when ozone or ozone and sulphur dioxide, nitrogen dioxide, or carbon monoxide were considered in a multipollutant model, however, the major association was still with PM₁₀. This result does not agree with the results of many Canadian studies in which the strongest associations were seen with gaseous pollutants and not respirable particulates.

Krewski et al. (2000)

The Health Effects Institute with support from the US EPA, industry, the US Congress, and other stakeholders, appointed an Expert Panel to perform a reanalysis of two major human epidemiology studies: (1) the Six Cities Study as reported by Dockery and co-workers in 1993 and (2) the American Cancer Society Study as reported by Pope and co-workers in 1995. Both of these studies estimated "that annual average all-cause mortality increased in association with an increase in fine particles (all particles less than 2.5 μ m in median aerodynamic diameter [PM_{2.5}])." Both were prospective cohort studies. The Six Cities Study followed a cohort of 8,111 adults in the Northeast and Midwest for 14-16 years. The American Cancer Society study followed 552,138 adults in 154 US cities for seven years.

Both of these studies were intensely scrutinized when the US EPA used the results to develop new National Ambient Air Quality Standards for PM_{2.5}. Neither group would release the data to

the public for review and evaluation, because they had collected it with guarantees of confidentiality for the participants. However, both studies were generated using federal funding, and the parties who wished to evaluate them insisted that independent reviews were required. To address this issue, both Harvard University and the American Cancer Society agreed to allow the Health Effects Institute to organize an independent reanalysis of the results.

The reanalysis team was headed by Dr. Daniel Krewski of the University of Ottawa. Primary authors of the reanalysis included Richard Burnett of Health Canada and five other Canadian researchers. The entire reanalysis team included 28 Canadian researchers.

The reanalysis team performed a large number of analyses that "included a set of new variables, all the gaseous copollutants, and the first attempts to apply spatial analytic methods to test the validity of the data and the results from the Six Cities Study and the ACS Study. Overall, the reanalysis confirmed many of the conclusions of the two original studies. In addition, however, the reanalyses extended the results.

In particular, the "use of spatial analytic methods suggested that, when the analyses controlled for correlations among cities located near one another, the associations between mortality and fine particles or sulfate remained but diminished." Also, "an association between sulfur dioxide and mortality was observed and persisted when other possible confounding variables were included; furthermore, when sulfur dioxide was included in models with fine particles or sulfate, the associations between these pollutants (fine particles and sulfate) and mortality diminished.

The final conclusion of the report was stated as follows:

In conclusion, the Reanalysis Team interpreted their findings to suggest that increased relative risk of "mortality may be attributed to more than one component of the complex mix of ambient air pollutants in urban areas in the United States." The Review Panel concurs. In the alternative analyses of the ACS Study cohort data, the Reanalysis Team identified relatively robust associations of mortality with fine particles, sulfate, and sulfur dioxide, and they tested these associations in nearly every possible manner within the limits of the datasets. Further investigations of these issues will enhance our understanding of the effect of combustion-source air pollutants (e.g, fine particles, sulfate, and sulfur dioxide) on public health.

5.11 Discussion of Prevalence and Causes of Asthma and Other Respiratory Diseases

Respiratory diseases in general and childhood asthma, in particular, are quite common diseases with multiple causes, which have been well studied.

5.11.1.1 Prevalence of Asthma

Asthma is known to be caused by many different stimuli. According to the National Academy of Sciences (1993), the prevalence of asthma has been on the rise nationally. The prevalence rates in the total U.S. population in 1991 for allergic diseases were 15-20% for allergic rhinitis (hay fever), 10-12% for sinusitis, and 8-11% for asthma.

According to Statistics Canada (1996-1997), the prevalence of asthma in New Brunswick is 6.6%, which is lower than the prevalence rates in all other provinces (7.1% to 8.8%) except for Newfoundland, where the prevalence rate is 6.0%. According to Asthma Society of Canada (1999), 10-20% of children in Canada are reported to have asthma.

Mannino et al. (1998) reported that the rate of self-reported asthma has increased steadily in the United States from 1980 to 1994. For all age groups, the rates have increased from 3.7% in 1980 to 5.4% in 1994. In children 5-14 years of age, the rates increased from 4.3% in 1980 to 7.4% in 1994. They report that asthma is one of the most common chronic diseases in the United States.

The National Academy of Sciences (2000) specifically studied asthma and found that the prevalence in 1993-1994 for the U.S. was 5% in males and 5.5% in females. Regional data show that asthma prevalence rates are higher in the Northeast and lowest in the Midwest and South.

The Centers for Disease Control (2001) reported in 2001 that the 1995 prevalence rate for asthma in the United States was 6% and the prevalence rate for children under 18 years of age was 7%.

According to EPA (2001a), about 17 million people of all age, race, and nationality have asthma. Nearly 1 in 13 of school-age children (4.8 million) has asthma.

According to the American Lung Association (2001a), asthma is the sixth-ranking chronic condition in the nation, and, in 1998, 26.3 people in the United States were estimated to have asthma. 1 in 13 children suffer from asthma nationally.

According to Anderson et al. (1987), 25% of 8,806 16-year-olds in England, Scotland, and Wales had asthma or wheezing in the past. There was a positive association between asthma or wheezing and other allergic conditions, such as eczema and allergic rhinitis (hayfever).

Prevalence rates for asthma were summarized by Cookson (1987). In Western countries, asthma rates for children were approximately 5% and rates for asthma or wheezing were approximately 10%. In Western countries, asthma rates for adults were approximately 3-5% and rates for asthma or wheezing were approximately 9%.

Evans et al. (1987) reported the asthma and wheezing prevalence rate for the United States for the years 1976-1980 as 11%.

Samet et al. (1988) studied the prevalence of asthma in over 2,000 Hispanic children in New Mexico and found that the rate of physician-diagnosed asthma was 7% in less than 6-year-old boys, 4% in 6-12 year-olds, and 11% in 13-17 year-olds. He concluded that these rates were similar to prevalence rates throughout the United States.

In conclusion, asthma is very common in Saint John, New Brunswick, and in Canada and the United States in general. The fact that some people living in Saint John are reporting that they and

their children have asthma is not unexpected and does not in any way point to emissions from the Coleson Cove Generating Plant as a causative factor.

5.11.1.2 Prevalence of Other Respiratory Diseases

Respiratory symptoms and illnesses are common in children. Schenker et al. (1983) summarized the prevalence rates in western Pennsylvania for over 4,000 children. For males aged 5-9 years the rates were as follows:

Disease	Prevalence Rate
Chronic cough	8%
Chronic phlegm	5%
Persistent Wheeze	10%
Asthma	5%
Hayfever	3%
Pneumonia	13%
Bronchitis	24%
Severe Chest Illness	11%
Chest Illness >3 days	13%

Risk factors included environmental tobacco smoke, parental history of allergy or respiratory illness, and lower socioeconomic status.

Samet et al. (1988) also summarized the prevalence rates for respiratory diseases in children. Out of over 2,000 Hispanic children studied in three age groups, the following were the results.

Disease	Prevalance Rate	
	Males	Females
Chronic cough	2-11%	4-11%
Chronic phlegm	4-8%	3-7%
Persistent wheeze	7-13%	3-5%
Asthma*, ever	4-11%	3-5%
Asthma*, current	3-8%	1-4%
Asthmatic bronchitis	3-4%	1-5%
Hay fever	5-20%	3-17%

* Physician-diagnosed

5.11.1.3 Causes of Asthma

According to the National Academy of Sciences (1993) asthma is an allergic disease: "Three common diseases are clearly related to exposure to indoor allergens: asthma, rhinitis (hay fever) and allergic skin conditions (i.e., eczema and urticaria)." Exposure to allergens, such as dust mites, animal dander, molds and pollen are major factors in the causation of asthma. 90% of children and 70% of young adults with asthma have clearly been shown to be allergic to specific allergens, such as house dust mite, grasses and weeds, animal danders, molds and cockroaches.

Biological sources of allergens that can cause asthma include: dust mites, spiders, cockroaches, crickets, flies, beetles, fleas, moths, midges, cats, dogs, ferrets, skunks, horses, rabbits, pigs, fungi, pollens, mice, rats, gerbils, and guinea pigs (NAS, 1993).

Environmental tobacco smoke can also promote the development of allergic diseases, such as asthma. Asthma is not caused by nonallergic dust, such as wind blown dust derived from native soils or respirable particulate matter. In fact, the only mention of air pollution as a factor in the development of asthma in the NAS (1993) report are statements that nonallergic factors, such as air pollution (ozone) "may modify the role of allergens in the disease process."

Asthma is also genetic in nature according to the National Academy of Sciences (1993). If neither parent has a history of allergy, a child will have a 0-19% chance of developing asthma. However, if one parent has a history of allergy, the probability of the child developing asthma is 31-58%. This probability increases to 60-100% if both parents have allergic disease. This fact was recently reiterated by the Academy (NAS, 2000): "There exists a substantial long-standing body of evidence that predisposition to asthma represents an inheritable phenomenon."

The National Academy of Sciences (2000) recently reported that asthma appears to be present in two forms: allergic asthma and nonallergic asthma. In the former case, exposure to specific allergens, such as dust mites and fungi, is required to cause the symptoms of asthma. The development of the allergic response may be influenced by respiratory infections and passive exposure to environmental tobacco smoke. Once asthma is present, other exposures in addition to exposure to allergens can cause asthma symptoms, such as airborne irritants, infections, and exercise. In case of nonallergic asthma, allergic responses cannot be demonstrated, and the cause of the symptoms is unclear. However, the NAS (2000) noted that approximately 80% of asthma in children is allergic asthma that requires exposure to specific, well-defined allergens.

The National Academy of Sciences (2000) summarized the agents that can cause or exacerbate asthma. They have concluded that the following exposures can cause the development of asthma: house dust mite, environmental tobacco smoke, and cockroach, respiratory syncytial virus. They have concluded that the following exposures exacerbate asthma in sensitive individuals: cats, cockroaches, house dust mites, environmental tobacco smoke, dogs, fungi or molds, rhinovirus, indoor nitrogen oxides, domestic birds, bacteria (*chlamydia pneumoniae* and *mycoplasma pneumoniae*), respiratory syncytial virus, formaldehyde, fragrances, ozone, respirable particulate matter (outdoor dust less than 10 microns in diameter), and sulfur dioxide.

The Centers for Disease Control (1999) have stated that people with asthma are sensitive to a variety of triggers for breathing problems. These triggers, which do not cause asthma, but may trigger the onset of asthmatic symptoms, include dust, tobacco smoke, cockroaches and some chemicals.

EPA (2001a) has stated that asthma attacks can be triggered by allergens, irritants and infections. Viral infections are the leading cause of acute asthma attacks. Bacterial sinusitis can also cause asthma attacks. Allergen triggers include cockroaches, dust mites, molds, pets and other animals with fur or feathers, and tree, grass, and weed pollen. Irritant triggers include cold air, second

hand cigarette smoke, ozone, industrial chemicals, perfume, paint and gasoline fumes, and combustion by-products. EPA (2001a) concluded that since people spend up to 90% of their time indoors, exposure to allergens and irritants in the home may "play a significant role in triggering asthma episodes."

The American Lung Association (2001) has reported that in people with asthma, many exposures can cause the "asthma symptoms to flare" including: air pollution (tobacco smoke, traffic fumes, high ozone levels), allergies (pollen, mold, animal dander, food), emotions (fear, crying, laughing), exercise (overexertion, exposure to cold air), household irritants (dust, cleaning products, perfume), illnesses (respiratory infections, colds), medications (some over-the-counter products, such as aspirin, ibuprofen, naproxen, ketoprofen), and weather (wind, cold air).

NAS (2000) reported that outdoor air pollution, such as ambient respirable particulate matter (outdoor dust less than 10 microns in diameter), "does not appear to play an important role in the increase in asthma in the United States since pollutant levels have been decreasing during the time of asthma increase."

Stieb et al. (2000a) in a study of cardiorespiratory episodes among patients visiting emergency departments in Saint John, New Brunswick found an "unexpectedly high proportion of ED [emergency department] visitors for respiratory conditions who were either daily smokers or who reported a regular smoker in the household, relative to results reported elsewhere for asthma." Also, they found that the most common primary trigger of respiratory disease episodes was viral upper respiratory tract infection. This fact was consistent with other published literature on asthma. Thus, smoking and exposure to environmental tobacco smoke and viral infections are important causative factors in triggering asthma symptoms that require hospitalization.

Greenberger and Patterson (1987) showed that an allergic response to the antigens from the fungus *Aspergillus fumigatus* can cause asthma and other respiratory diseases.

Woolcock (1987) concluded that there is a positive association between asthma and parental history of asthma, history of bronchitis before the age of 2, and positive allergic skin tests to common allergens.

Tager et al. (1987) summarized the literature and concluded that the risks factors that have been shown in epidemiology studies to be associated with the occurrence of asthma in children are being male, having a parental history of asthma, having allergic responses (allergic rhinitis or hayfever, positive skin test to known allergens or detectable serum antibodies to known allergens), and having low socioeconomic status.

Reed and Swanson (1987) discussed the causes of asthma and concluded that 60-80% of people with asthma have allergen-induced health episodes. Typical allergens include tree, grass, and weed pollen, molds, insects, housedust mites, pets, cockroaches, molds and bacteria in humidifiers and air conditioners, and others. Regarding trees, grasses and weeds, their work demonstrated that allergic responses are not restricted to pollens. The allergic agents can be found in other plant parts and even plant detritus present in soil.

Ware et al. (1984) studied passive smoking and childhood respiratory disease. The report that most studies of children exposed to environmental cigarette smoke have found increases in respiratory illness, respiratory symptoms, and reductions in indices of lung functioning. In this study of over 10,000 children in six cities, environmental tobacco smoke was associated with increase in several respiratory illnesses and indices investigated (doctor-diagnosed respiratory illness before the age of 2, history of bronchitis, respiratory illness last year, bronchitis, cough, wheeze, and lower respiratory index). Suggestive evidence was presented that gas stoves are associated with increased respiratory illnesses in children.

Weiss et al. (1980) studied early-life risk factors for the development of adult obstructive airway disease in 650 children. Parental cigarette smoking was associated with persistent wheeze, and children with persistent wheezing were more likely to report cough and phlegm, have a history of asthma, have hay fever, or have past hospitalizations for respiratory illness.

Coultas (1998) summarized the scientific literature on passive smoking (environmental tobacco smoke) and asthma and chronic obstructive pulmonary disease (COPD). According to the literature, the onset of asthma during infancy and childhood may be associated with exposure to environmental tobacco smoke. In addition, growing evidence suggests that exposure to environmental tobacco smoke is a risk factor for adult onset asthma and chronic obstructive pulmonary disease.

In conclusion, asthma is multi-causal and has not been shown to be caused by air pollution. Air pollution is generally recognized to be one of many factors that can trigger asthmatic symptoms, but there are so many other factors that have been shown to trigger asthma within the City of Saint John, as well as within people's homes, that one cannot conclude that emissions from the Coleson Cove Generating Station have caused or exacerbated asthmatic conditions in Saint John.

5.12 Discussion of Susceptible Populations

According to New Brunswick Health and Wellness (2002), children, elderly people, and people with cardiac and respiratory diseases are more susceptible to the effects of air pollution on health than the average adult. One of the reasons that children are stated to be more sensitive than adults is that they spend more time outdoors compared to adults. In Saint John in the summer, adults only spend 10% of their time outdoors, but children spend 19% of their time outdoors. They also have higher breathing rates than do adults.

Raizenne et al. (1998) reported that children with asthma or asthma-like symptoms are at the highest risk of developing short term and long term health effects due to air pollutants. The authors also discussed indoor air pollutants and their effect on children's health: "Environmental tobacco smoke is a well-recognized persistent indoor air contaminant with adverse effects in children of all ages. Uncontrolled moisture in the indoor environment is increasingly recognized to significantly increase the risk of respiratory morbidity in children."

In its "Air Quality Criteria for Particulate Matter," the United States Environmental Protection Agency (EPA, 2001b) has concluded that adverse health effects in children is an important area of concern. However, EPA has noted: "Unfortunately, relatively little is known about the relationship of PM to the most serious health endpoints (low birth weight, preterm birth, neonatal and infant mortality, emergency hospital admissions and mortality in older children). Also, little is yet known about involvement of PM exposure in the progress from less serious childhood conditions, such as asthma and respiratory symptoms, to more serious disease endpoints later in life."

This risk assessment specifically takes into account the possibility that children may be more sensitive to air pollution than adults. First, the differences in inhalation rates and the lower body weights are explicitly incorporated into the risk assessment calculations. Second, the tolerable daily intake (TDI) or reference dose (RfD) values that are used to evaluate the toxicity associated with a certain estimated chemical dose level have been derived by governmental health agencies specifically to be protective of sensitive members of the population, which includes the developing fetus, children, the elderly, and people with impaired health status. The TDIs and RfDs assume that sensitive members of the population are a factor of ten times more sensitive than a healthy adult.

The risk assessment also takes into account the possibility that elderly adults or adults with pre-existing cardiorespiratory disease may be more sensitive to air pollution than normal adults. It does this by assuming that the exposed adult is an active person who spends 100% of their time outdoors breathing the estimated airborne chemical concentrations. In reality, elderly adults and adults with cardiorespiratory diseases will spend only a small fraction of their time outdoors. Since indoor air concentrations of ambient air pollutants are much less inside of buildings than outside, the risk assessment overestimates the exposures to elderly adults and adults with pre-existing diseases. Secondly, as noted above, the TDIs and RfDs that are used in the risk assessment are defined to be protective of elderly adults and adults with cardiorespiratory diseases.

5.13 Comparison of Impacts of Coleson Cove Emissions to Background Conditions

5.13.1 Air

Limited data are available on metal levels in airborne particulate matter in Saint John. As can be seen in Table 5-37, the levels of metals estimated in Saint John air from emissions of the Coleson Cove Generating Plant are far less than the ambient levels for most metals. For these metals, Coleson Cove is an insignificant source of these metals in ambient air. For nickel and vanadium, the contribution of Coleson Cove is estimated to be somewhat higher. In the Existing Condition - Fuel Oil #6 - High Load Scenario assuming operation for 30 years, the estimated emissions of nickel from Coleson Cove comprise 5% of the measured ambient concentration. In the Refurbishment Project - Orimulsion® - High Load Scenario assuming operation for 30 years, the estimated emissions of nickel from Coleson Cove comprise 2% of the measured ambient concentration. In the Existing Condition - Fuel Oil #6 - High Load Scenario assuming operation for 30 years, the estimated emissions of vanadium from Coleson Cove comprise 7% of the measured ambient concentration. In the Refurbishment Project - Orimulsion® - High Load Scenario assuming operation for 30 years, the estimated emissions from Coleson Cove comprise 1.5% of

the measured ambient concentration. Thus, levels of all metals would be predicted to fall with the refurbishment project. For most metals, the improvement in air quality would probably not be measurable. However, for nickel and vanadium, measurable improvements may be seen.

A discussion of criteria pollutants can be found in section 5.15.

5.13.2 Soil

The levels of metals estimated to be present in the soil of Saint John residents after 30 years of operation of the Coleson Cove Generating Plant assumed to be operating at full load were compared to the typical levels of these metals in Canadian soils in Table 5-39. The Canadian Council of Ministers of the Environment document entitled Canadian Environmental Quality Guidelines (CCME, 1999) was consulted, and typical levels were found for mercury, arsenic, cadmium, lead, nickel, and vanadium. In the absence of data for beryllium and chromium in CCME (1999), a United States government document was consulted (ATSDR, 1992).

The levels of metals estimated to be present in soils from emissions from the plant range from 0.0004% to 5% of typical levels for the Heavy Fuel Oil Fuel scenario and from 0.00004% to 1% of typical levels for the Orimulsion Fuel scenario. Thus, the Coleson Cove Generating Plant is not a major source of the metals in local soils. Nonetheless, the contribution of metals to soils will be lessened with the Coleson Cove Refurbishment Project.

5.13.3 Fish Tissue

The maximum level of mercury present in fish in the nearest waterbodies assuming that the CCGS operated at full capacity for 24 hours a day for 365 days a year for 30 years of operation is estimated to be 0.03 mg/kg for the Existing Operation - Fuel Oil #6 - High Load Scenario, and 0.006 mg/kg for the Refurbishment Project - Orimulsion® - High Load Scenario, and 0.00009 mg/kg for the Natural Gas - High Load Scenario. These estimated levels of mercury in fish due to emissions from the CCGS can be compared to the average level of mercury in fish in New Brunswick.

In 1994, the New Brunswick Departments of Environment and Natural Resources and Energy performed a survey of brook trout in 35 lakes throughout the Province (New Brunswick Department of Environment, 1997). A total of 108 fish were collected. The average level of mercury in the New Brunswick brook trout fish was 0.18 mg/kg. The average level in brook trout taken from lakes in Charlotte, King and St. John counties was 0.38 mg/kg. Other fish species were also collected from some of the lakes, including brown trout, pickerel, yellow perch, landlocked salmon, small mouth bass, and white sucker. The average level of mercury of 18 fish samples of other species in New Brunswick was 0.55 mg/kg and 0.44 mg/kg for fish from lakes in Charlotte, King and St. John counties.

Results from previous studies were also reported. The average mercury concentration found in 1987 in brook trout in Sissons Branch and Trousers Lake was 0.43 mg/kg and 0.25 mg/kg. The average mercury concentration found in 1987 in landlocked salmon in Sissons Branch and Trousers Lake was 0.78 mg/kg and 0.33 mg/kg.

In 1977, smallmouth bass were analyzed from two New Brunswick lakes and mercury levels were 0.95 and 1.13 mg/kg in the two lakes. Mercury in landlocked salmon from two lakes were 0.11 and 0.25 mg/kg. Mercury in lake trout from one lake was 0.9 mg/kg and in striped bass from one lake was 0.11 mg/kg. Brook trout were analyzed in 1970 from three water bodies in New Brunswick, and average levels of mercury were 0.08, 0.12 and 0.13 mg/kg.

A major conclusion drawn by the New Brunswick Departments of Environment and Natural Resources and Energy from their 1994 study was that brook trout have higher mercury concentrations as they age. All but one of the fish with lengths less than 30 cm had mercury levels less than 0.5 mg/kg, which was reported as the level that does not represent a health hazard when eaten daily for extended periods of time. The Departments also concluded that "the levels of mercury in brook trout in New Brunswick have not changed appreciably in the last 24 years."

The estimated level of mercury in fish due to emissions from the CCGS are small compared to the levels of mercury that have been measured in fish in New Brunswick lakes. Specifically, the emissions from CCGS assuming 30 years of emissions with the Existing Operation - Fuel Oil #6 - High Load Scenario are estimated to explain only 8% of the mercury in fish in the nearest waterbodies, using the data from brook trout from Charlotte, King and St. John counties as the point of comparison. With the Refurbishment Project - Orimulsion® - High Load Scenario, this fraction drops to 1.5%. In the Natural Gas - High Load Scenario, the CCGS emissions would contribute less than 1% of the mercury in fish. The contribution of CCGS emissions to fish tissue mercury levels in waterbodies more distant would be expected to be even less, because the deposition rates are lower than they are in the nearest watershed.

Comparisons to the average level of mercury in other species from these counties are very similar.

The total amount of mercury estimated to enter the nearest waterbodies from CCGS emissions was estimated to be 5.3 grams per year for the Existing Operation - Fuel Oil #6 - High Load Scenario and 0.9 grams per year for the Refurbishment Project - Orimulsion® - High Load Scenario. For the Natural Gas - High Load Scenario, the total amount of mercury estimated to enter the nearest waterbodies from CCGS emissions was estimated to be 0.02 grams per year. For comparison, the amount of mercury in an average clinical thermometer ranges from 0.6 grams to 2.3 grams according to US EPA (1997b).

Thus, the Coleson Cove Generating Plant is not a major source of the mercury in local fish. Nonetheless, the contribution of mercury to local fish will be lessened with the Coleson Cove Refurbishment Project.

5.14 Conclusions About Effects of Coleson Cove on Cardiorespiratory Health Outcomes

In conclusion, even though the decrease in air pollutant concentrations that are estimated to occur when the Coleson Cove facility is refurbished are expected to have a beneficial effect on human health, there is no way to quantitatively predict the number of cases of each health outcome that

will be prevented. This human health risk assessment has predicted the differences in carcinogenic and noncarcinogenic health risk for selected metals, polycyclic aromatic hydrocarbons, and volatile organic chemicals, because there are standard risk assessment methodologies developed by government health protection agencies to do so. Such methods have been widely used for environmental decision making for over 25 years. However, no such methods exist for estimating the number of hospitalizations for asthma, chronic obstructive pulmonary disease, or cardiovascular disease or other similar health outcomes. Based on the accompanying review of recent scientific studies, one can make only qualitative conclusions about the improvement of the health status of residents of Saint John expected with the refurbishment.

Based on a review of all of the studies performed in Canada and the United States, it can be stated that reductions in ambient concentrations of one or more of the following air pollutants (ozone, sulphur dioxide, nitrogen dioxide, carbon monoxide, and respirable particulate matter [$PM_{2.5}$ and PM_{10}]) would be expected to confer health benefits to residents of Saint John. These benefits would be associated with hospitalizations for asthma and other respiratory diseases, hospitalizations for cardiac diseases, and mortality from cardiorespiratory diseases. However, this risk assessment specifically concerns the emissions from a single source of air pollution in the area. Therefore, reductions in emissions from the Coleson Cove Generating Station would only change the ambient concentrations of air pollutants in Saint John and confer health benefits to the residents of Saint John if the facility were a major source of ambient air pollution in the area.

In fact, the Coleson Cove facility is not a major source of air pollution in the city of Saint John. Assuming that the facility were operated at full load every day of the year using fuel oil #6, the estimated concentrations of respirable particulate matter [$PM_{2.5}$ and PM_{10}] have been estimated to be approximately 0.1% of the measured air pollutant concentrations in the City of Saint John. Assuming that the CCGS operated every day at full capacity using fuel oil #6 as fuel, the CCGS would be estimated to contribute approximately 8% to the annual average sulphur dioxide concentration and approximately 3% to the nitrogen oxide concentration.

These results are consistent with the results presented by Brook et al. (1995). Thus, the reductions in concentrations of sulphur dioxide, nitrogen dioxide, and respirable particulate matter expected after the refurbishment project is complete may not be measurable, even though the emissions from Coleson Cove will be reduced. This is because the refurbishment project will have no affect on the major sources of air pollutants that affect the City of Saint John.

In addition, although it may be reasonable to assume that some asthma and cardiorespiratory symptoms and disease outcomes are caused by air pollution, these diseases are multi-causal. As noted in Section 5.11.1.3, asthma researchers believe that exposure to specific allergens, such as those associated with pollens, fungi, dust mites, insects and pet dander, and environmental tobacco smoke are the major causes of asthma. Of course, outdoor air pollution is thought to exacerbate asthma symptoms, as are other environmental factors, such as exposure to fragrances and household cleaning products, viral infections, exercise, and cold air. Similarly, chronic obstructive pulmonary disease is associated with smoking in addition to air pollution. Thus, reduction of emissions of sulphur dioxide, nitrogen dioxide, respirable particulate matter, and specific chemicals associated with the particulate matter will reduce some of the agents that can

cause or exacerbate cardiorespiratory diseases. This is a benefit to the public health. However, the causation of these diseases and the triggering of their acute symptoms are extremely complex phenomena that cannot be explained in totality. Thus, decreases in the emissions from Coleson Cove Generating Station may or may not translate into a measurable decrease in the number of new cases of cardiorespiratory diseases, the number of cardiorespiratory hospitalizations, the number of asthma attacks in children, or any other such similar health outcomes. The causation of these diseases is too complex to make any such quantitative predictions. Nonetheless, reductions in the emissions of criteria pollutants is a positive benefit to health.

5.15 Risk Characterization of Criteria Pollutants

Estimated annual average concentrations of sulphur dioxide, nitrogen oxides, and respirable particulate matter are compared to the measured levels in Saint John in Table 5-38. The CCGS is not a major source of sulphur dioxide, nitrogen dioxide, PM_{10} , or $PM_{2.5}$ in the City of Saint John, nor would it be if the refurbishment project were executed. For instance, assuming that the average concentration of sulphur dioxide in Saint John is 18 micrograms per cubic meter of air (ug/m^3), which is the average of the Forest Hills and the Prince William Street measurements from 1995-2000 and assuming that the CCGS operated at maximum capacity for 24 hours and day for 365 days a year, the CCGS would be estimated to contribute approximately 8% to the annual average concentration. Under actual plant operation, the fraction of the annual average sulphur dioxide concentration in the City of Saint John is much less than 8%.

Similarly, assuming that the average concentration of nitrogen oxides in Saint John is $14.5 ug/m^3$, which is the average of the Forest Hills and the Prince William Street measurements from 1995-2000 and assuming that the CCGS operated at maximum capacity for 24 hours and day for 365 days a year, the CCGS would be estimated to contribute approximately 3% to the annual average concentration. Under actual plant operation, the fraction of the annual average nitrogen dioxide concentration in the City of Saint John is much less than 3%.

Also, assuming that the average concentration of PM_{10} and $PM_{2.5}$ in Saint John is 14 and $9 ug/m^3$, which are the averages of the Forest Hills and the Prince William Street measurements from 1995-2000 and assuming that the CCGS operated at maximum capacity for 24 hours and day for 365 days a year, the CCGS would be estimated to contribute 0.1% to the annual average concentration for both PM_{10} and $PM_{2.5}$. Under actual plant operation, the fraction of the annual average nitrogen dioxide concentration in the City of Saint John is much less than 0.1%.

Neither estimated concentrations resulting from the CCGS nor the existing ambient concentrations that result primarily from sources other than the CCGS exceed standards derived to protect human health. As discussed in the Air Quality Report (JWEL, 2002), the standards for the criteria pollutants are $30 ug/m^3$ for sulphur dioxide (annual average), $100 ug/m^3$ for nitrogen dioxide (annual average), $70 ug/m^3$ for total particulate matter (annual average), $50 ug/m^3$ for PM_{10} (24-hour average), and $30 ug/m^3$ for $PM_{2.5}$ (24-hour average). For comparison the respirable particulate matter standards in the United States are $50 ug/m^3$ for PM_{10} (annual average), and $15 ug/m^3$ for $PM_{2.5}$ (annual average). None of these standards is exceeded by the estimated criteria

pollutant concentrations resulting from emissions of the CCGS or for the ambient conditions in the City of Saint John.

All of the ambient criteria pollutant concentrations are less than their respective standards, but some are within a factor two or less of the standard. For instance, the standard for sulphur dioxide is 30 ug/m^3 , which is a Maximum Desirable Level as defined by the Working Group on Air Quality Objectives and Guidelines. The ambient level in Saint John is roughly two-fold lower than the standard. However it is four-fold lower than the level that has been defined as the Maximum Acceptable Level. The standard for nitrogen dioxide is 100 ug/m^3 for nitrogen dioxide, which is a Maximum Acceptable Level as defined by the Working Group on Air Quality Objectives and Guidelines. The ambient levels in Saint John are a factor of seven below this level and a factor of four lower than the level defined as the Maximum Desirable Level. Both PM_{10} and $\text{PM}_{2.5}$ ambient levels are 3 times lower than the standards.

Because ambient concentrations of these criteria pollutants are only factors of two to seven below the governmental standards, it is desirable to reduce significant sources of emissions of criteria pollutants in an attempt to bring ambient levels down. Even though the major sources of criteria pollutants include long range transport from the eastern United States and emissions from industries within the city limits, the CCGS is responsible for some, albeit small, fraction of the ambient concentrations in the City of Saint John.

If the refurbishment project were executed, the contribution of the CCGS to the concentrations in Saint John would drop, so the ambient concentrations in Saint John would also drop assuming that no other sources were increased. This estimated drop the concentrations of criteria pollutants is a health benefit.

However, since the CCGS is not a major source of these criteria pollutants in the City of Saint John, the predicted decreases in concentrations of criteria pollutants are modest and may be difficult to measure. For instance, assuming that the average concentration of sulphur dioxide in Saint John is 18 micrograms per cubic meter of air (ug/m^3), as noted above, the average concentration is estimated to drop from 18 to 17.5 ug/m^3 if the refurbishment project were executed and assuming maximum capacity operations for all scenarios. The estimated additional improvement in air quality assuming that the CCGS used natural gas as fuel would result in an estimated concentration of 16.5 ug/m^3 .

Similarly, assuming that the average concentration of nitrogen oxides in Saint John is 14.5 ug/m^3 , as noted above, the average concentration is estimated to drop from 14.5 to 14.4 ug/m^3 if the refurbishment project were executed and assuming maximum capacity operations for all scenarios. The estimated additional improvement in air quality assuming that the CCGS used natural gas as fuel would result in an estimated concentration of 14.2 ug/m^3 .

For respirable particulate matter, the CCGS is estimated to contributed only 0.1% to the concentrations of PM_{10} and $\text{PM}_{2.5}$ in the City of Saint John. Thus, the estimated improvements in the concentrations assuming that the refurbishment project is executed are probably not measurable. PM_{10} is estimated to drop from 14 ug/m^3 to 13.99 ug/m^3 with the refurbishment and

assuming maximum capacity operations for all scenarios and to 13.98 ug/m^3 if it were assumed that natural gas were used as fuel. $\text{PM}_{2.5}$ is estimated to drop from 9 ug/m^3 to 8.995 ug/m^3 with the refurbishment and assuming maximum capacity operations for all scenarios and to 8.988 ug/m^3 if it were assumed that natural gas were used as fuel.

Table 5-1
Summary of Potential Hazard Indices for Fuel Oil High Load Conditions
Resident at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child				Adult			
	Soil	Water	Air	HI	Soil	Water	Air	HI
Mercury, elemental	NC	NC	4.16E-11	4.156E-11	NC	NC	3.55E-11	3.551E-11
Mercury, methyl	1.31E-05	9.33E-07	NC	2.39E-06	2.43E-07	3.25E-07	NC	1.42E-06
Mercury, divalent	7.36E-04	3.68E-06	2.72E-09	2.38E-05	1.37E-05	1.28E-06	NC	1.40E-05
1,4-Dichlorobenzene	2.00E-11	7.91E-10	8.86E-12	2.68E-08	3.71E-13	2.75E-10	7.57E-12	1.49E-08
Acenaphthene	2.42E-09	1.25E-09	9.96E-12	4.65E-08	4.49E-11	4.35E-10	8.51E-12	2.60E-08
Acenaphthylene	6.58E-08	2.12E-08	1.19E-13	3.07E-08	1.22E-09	7.38E-09	1.02E-13	1.84E-08
Anthracene	2.56E-10	1.69E-11	1.15E-13	2.26E-10	4.75E-12	5.88E-12	9.83E-14	1.35E-10
Arsenic	1.38E-04	8.18E-04	1.49E-08	6.51E-04	2.56E-06	2.85E-04	1.27E-08	3.90E-04
Benzene	9.26E-11	1.81E-07	3.95E-09	2.98E-07	1.72E-12	6.29E-08	3.37E-09	1.69E-07
Benzo(a)anthracene	1.25E-07	4.22E-09	7.10E-12	1.50E-08	2.33E-09	1.47E-09	6.07E-12	8.99E-09
Benzo(a)pyrene	2.77E-08	9.62E-10	1.95E-12	1.94E-08	5.15E-10	3.35E-10	1.66E-12	1.16E-08
Benzo(b)fluoranthene	9.06E-08	1.65E-09	5.42E-12	1.42E-08	1.68E-09	5.74E-10	4.63E-12	8.48E-09
Benzo(k)fluoranthene	2.87E-07	6.04E-09	5.42E-12	6.55E-08	5.33E-09	2.10E-09	4.63E-12	3.93E-08
Benzo(g,h,i)perylene	5.87E-07	1.89E-07	1.60E-11	6.24E-08	1.09E-08	6.58E-08	1.36E-11	3.72E-08
Beryllium	1.89E-05	4.89E-06	2.85E-08	3.58E-06	3.50E-07	1.70E-06	2.43E-08	2.15E-06
Cadmium	3.55E-06	8.26E-06	1.48E-10	1.47E-05	6.59E-08	2.88E-06	1.27E-10	8.80E-06
Chromium	3.16E-06	7.50E-10	4.87E-12	2.26E-07	5.87E-08	2.61E-10	4.16E-12	1.36E-07
Chromium VI	1.17E-05	1.06E-04	7.30E-08	8.26E-05	2.18E-07	3.68E-05	6.24E-08	4.96E-05
Chrysene	1.08E-07	5.13E-09	4.22E-12	2.26E-07	2.00E-09	1.79E-09	3.60E-12	9.81E-09
Dibenzo(a,h)anthracene	1.86E-07	2.28E-09	2.95E-12	9.98E-08	3.46E-09	7.95E-10	2.52E-12	5.99E-08
Fluoranthene	3.74E-08	1.69E-09	3.42E-12	3.66E-09	6.95E-10	5.87E-10	2.92E-12	2.17E-09
Fluorene	5.56E-09	2.38E-08	3.16E-12	4.65E-09	1.03E-10	8.30E-09	2.70E-12	2.79E-09
Formaldehyde	4.19E-11	2.98E-07	5.18E-09	1.35E-06	7.78E-13	1.04E-07	4.43E-09	7.65E-07
Indeno(1,2,3-cd)pyrene	1.87E-07	1.18E-09	3.78E-12	1.28E-07	3.48E-09	4.12E-10	3.23E-12	7.67E-08
Lead	9.98E-03	2.32E-03	1.67E-07	2.07E-03	1.85E-04	8.07E-04	1.43E-07	1.24E-03
Naphthalene	3.14E-08	1.52E-07	2.00E-09	4.77E-06	5.83E-10	5.28E-08	1.71E-09	2.69E-06
Nickel	2.19E-03	5.86E-03	3.70E-04	4.88E-03	4.06E-05	2.04E-03	3.16E-04	2.93E-03
Phenanthrene	2.55E-10	7.28E-09	7.42E-11	9.40E-09	4.73E-12	2.54E-09	6.34E-11	5.63E-09
Pyrene	1.47E-07	4.75E-08	4.01E-12	6.88E-08	2.74E-09	1.65E-08	3.42E-12	4.12E-08
Toluene	8.79E-11	7.53E-08	1.56E-10	2.03E-07	1.63E-12	2.62E-08	1.33E-10	1.14E-07
Vanadium	2.46E-02	7.72E-02	1.54E-06	6.80E-02	4.56E-04	2.69E-02	1.32E-06	4.08E-02
Total HI	3.77E-02	8.63E-02	3.72E-04	7.57E-02	6.99E-04	3.00E-02	3.18E-04	4.54E-02

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-2

Summary of Potential Excess Lifetime Cancer Risk for Fuel Oil High Load Conditions
Resident at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child				Adult					
	Soil	Water	Air	Produce	Risk	Soil	Water	Air	Produce	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	9.46E-15	3.74E-13	4.17E-15	1.26E-11	1.30E-11	8.79E-16	6.51E-13	1.78E-14	3.52E-11	3.59E-11
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	5.84E-09	3.47E-08	8.58E-12	2.77E-08	6.83E-08	5.42E-10	6.05E-08	3.67E-11	8.31E-08	1.44E-07
Benzene	6.90E-16	1.35E-12	1.67E-14	2.22E-12	3.58E-12	6.41E-17	2.34E-12	7.13E-14	6.29E-12	8.71E-12
Benzo(a)anthracene	4.54E-11	1.61E-12	3.02E-15	5.79E-12	5.28E-11	4.21E-12	2.79E-12	1.29E-14	1.73E-11	2.43E-11
Benzo(a)pyrene	1.02E-10	3.71E-12	8.28E-15	7.62E-11	1.82E-10	9.46E-12	6.47E-12	3.54E-14	2.29E-10	2.44E-10
Benzo(b)fluoranthene	3.29E-11	6.33E-13	2.30E-15	5.54E-12	3.91E-11	3.06E-12	1.10E-12	9.84E-15	1.66E-11	2.08E-11
Benzo(k)fluoranthene	8.59E-12	2.03E-13	2.30E-16	2.54E-12	1.13E-11	7.98E-13	3.53E-13	9.84E-16	7.61E-12	8.76E-12
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	ND	ND	1.17E-13	ND	1.17E-13	ND	ND	4.99E-13	ND	4.99E-13
Cadmium	ND	ND	3.58E-13	ND	3.58E-13	ND	ND	1.53E-12	ND	1.53E-12
Chromium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	4.81E-11	ND	4.81E-11	ND	ND	2.06E-10	ND	2.06E-10
Chrysene	3.74E-13	1.90E-14	1.79E-17	6.30E-14	4.56E-13	3.48E-14	3.31E-14	7.66E-17	1.89E-13	2.57E-13
Dibenz(a,h)anthracene	6.45E-10	8.56E-12	1.25E-14	3.93E-10	1.05E-09	5.99E-11	1.49E-11	5.39E-14	1.18E-09	1.25E-09
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	4.00E-12	ND	4.00E-12	ND	ND	1.71E-11	ND	ND
Indeno(1,2,3-cd)pyrene	6.68E-11	4.52E-13	1.61E-15	5.04E-11	1.18E-10	6.20E-12	7.86E-13	6.86E-15	1.51E-10	1.58E-10
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	7.91E-10	ND	7.91E-10	ND	ND	3.38E-09	ND	3.38E-09
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total Risk	6.74E-09	3.47E-08	8.52E-10	2.82E-08	7.06E-08	6.25E-10	6.05E-08	3.64E-09	8.47E-08	1.49E-07

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-3
Summary of Potential Hazard Indices for Fuel Oil High Load Conditions
Resident at Saint John
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child				Adult					
	Soil	Water	Air	Produce	HI	Soil	Water	Air	Produce	HI
Mercury, elemental	NC	NC	1.87E-09	NC	1.87E-09	NC	NC	1.60E-09	NC	1.59787E-09
Mercury, methyl	4.14E-07	9.33E-07	NC	6.20E-08	1.41E-06	7.69E-09	3.25E-07	NC	3.67E-08	3.69E-07
Mercury, divalent	2.32E-05	3.68E-06	1.22E-07	8.26E-07	2.79E-05	4.32E-07	1.28E-06	1.04E-07	4.87E-07	2.30E-06
1,4-Dichlorobenzene	6.66E-13	7.91E-10	3.99E-10	8.91E-10	2.08E-09	1.24E-14	2.75E-10	3.41E-10	4.97E-10	1.11E-09
Acenaphthene	8.06E-11	1.25E-09	4.48E-10	1.55E-09	3.33E-09	1.50E-12	4.35E-10	3.83E-10	8.68E-10	1.69E-09
Acenaphthylene	1.56E-09	2.12E-08	5.17E-12	8.39E-10	2.36E-08	2.91E-11	7.38E-09	4.42E-12	5.03E-10	7.92E-09
Anthracene	8.51E-12	1.69E-11	5.18E-12	7.53E-12	3.81E-11	1.58E-13	5.88E-12	4.42E-12	4.50E-12	1.50E-11
Arsenic	2.20E-06	8.18E-04	5.36E-07	1.21E-05	8.33E-04	4.09E-08	2.85E-04	4.57E-07	7.23E-06	2.93E-04
Benzene	3.08E-12	1.81E-07	1.78E-07	9.91E-09	3.68E-07	5.73E-14	6.29E-08	1.52E-07	5.62E-09	2.20E-07
Benzo(a)anthracene	4.01E-09	4.22E-09	3.18E-10	4.10E-10	8.95E-09	7.44E-11	1.47E-09	2.71E-10	2.45E-10	2.06E-09
Benzo(a)pyrene	6.83E-10	9.62E-10	8.48E-11	4.77E-10	2.21E-09	1.27E-11	3.35E-10	7.24E-11	2.86E-10	7.06E-10
Benzo(b)fluoranthene	2.83E-09	1.65E-09	2.42E-10	3.82E-10	5.11E-09	5.26E-11	5.74E-10	2.07E-10	2.29E-10	1.06E-09
Benzo(k)fluoranthene	6.65E-09	6.04E-09	2.35E-10	3.82E-10	1.46E-08	1.24E-10	2.10E-09	2.00E-10	9.75E-10	3.40E-09
Benzo(g,h,i)perylene	1.40E-08	1.89E-07	6.92E-10	3.50E-09	2.07E-07	2.59E-10	6.58E-08	5.91E-10	2.09E-09	6.88E-08
Beryllium	3.02E-07	4.89E-06	1.02E-06	6.63E-08	6.28E-06	5.60E-09	1.70E-06	8.76E-07	3.98E-08	2.62E-06
Cadmium	5.67E-08	8.26E-06	5.34E-09	2.52E-07	8.57E-06	1.05E-09	2.88E-06	4.56E-09	1.51E-07	3.03E-06
Chromium	5.06E-08	7.50E-10	1.75E-10	4.16E-09	5.57E-08	9.40E-10	2.61E-10	1.50E-10	2.49E-09	3.84E-09
Chromium VI	1.87E-07	1.06E-04	2.63E-06	1.53E-06	1.10E-04	3.48E-09	3.68E-05	2.25E-06	9.21E-07	3.99E-05
Chrysene	3.30E-09	5.13E-09	1.88E-10	4.54E-10	9.07E-09	6.12E-11	1.79E-09	1.60E-10	2.71E-10	2.28E-09
Dibenzo(a,h)anthracene	2.99E-09	2.28E-09	1.07E-10	2.28E-09	7.66E-09	5.56E-11	7.95E-10	9.10E-11	1.37E-09	2.31E-09
Fluoranthene	1.24E-09	1.69E-09	1.54E-10	1.19E-10	3.20E-09	2.31E-11	5.87E-10	1.31E-10	7.05E-11	8.12E-10
Fluorene	1.81E-10	2.38E-08	1.42E-10	1.36E-10	2.43E-08	3.36E-12	8.30E-09	1.21E-10	8.13E-11	8.51E-09
Formaldehyde	1.39E-12	2.98E-07	2.33E-07	4.48E-08	5.76E-07	2.59E-14	1.04E-07	1.99E-07	2.55E-08	3.28E-07
Indeno(1,2,3-cd)pyrene	3.00E-09	1.18E-09	1.36E-10	4.40E-09	8.73E-09	5.58E-11	4.12E-10	1.16E-10	2.64E-09	3.22E-09
Lead	1.60E-04	2.32E-03	6.02E-06	3.73E-05	2.52E-03	2.97E-06	8.07E-04	5.14E-06	2.24E-05	8.37E-04
Naphthalene	1.05E-09	1.52E-07	8.99E-08	1.59E-07	4.01E-07	1.94E-11	5.28E-08	7.68E-08	8.95E-08	2.19E-07
Nickel	3.50E-05	5.86E-03	1.33E-02	8.97E-05	1.93E-02	6.49E-07	2.04E-03	1.14E-02	5.38E-05	1.35E-02
Phenanthrene	8.48E-12	7.28E-09	3.34E-09	3.13E-10	1.09E-08	1.57E-13	2.54E-09	2.85E-09	1.87E-10	5.58E-09
Pyrene	3.51E-09	4.75E-08	1.74E-10	1.88E-09	5.31E-08	6.51E-11	1.65E-08	1.49E-10	1.13E-09	1.79E-08
Toluene	2.93E-12	7.53E-08	7.01E-09	6.75E-09	8.91E-08	5.44E-14	2.62E-08	5.99E-09	3.79E-09	3.60E-08
Vanadium	3.93E-04	7.72E-02	5.55E-05	1.26E-03	7.89E-02	7.30E-06	2.69E-02	4.74E-05	7.54E-04	2.77E-02
Total HI	6.14E-04	8.63E-02	1.34E-02	1.40E-03	1.02E-01	1.14E-05	3.00E-02	1.14E-02	8.39E-04	4.23E-02

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-4
Summary of Potential Excess Lifetime Cancer Risk for Fuel Oil High Load Conditions
Resident at Saint John
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child			Adult			Risk
	Soil	Water	Air	Soil	Water	Air	
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	3.15E-16	3.74E-13	1.87E-13	2.93E-17	6.51E-13	8.01E-13	2.62E-12
Acenaphthene	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND
Anthracene	9.33E-11	3.47E-08	3.09E-10	8.67E-12	6.05E-08	1.32E-09	6.33E-08
Arsenic	2.30E-17	1.35E-12	7.51E-13	2.13E-18	2.34E-12	3.21E-12	5.76E-12
Benzene	1.45E-12	1.61E-12	1.35E-13	1.35E-13	2.79E-12	5.77E-13	3.98E-12
Benzo(a)anthracene	2.51E-12	3.71E-12	3.60E-13	2.33E-13	6.47E-12	1.54E-12	1.39E-11
Benzo(a)pyrene	1.03E-12	6.33E-13	1.03E-13	9.56E-14	1.10E-12	4.39E-13	2.08E-12
Benzo(b)fluoranthene	1.99E-13	2.03E-13	9.97E-15	1.85E-14	3.53E-13	4.26E-14	6.03E-13
Benzo(k)fluoranthene	ND	ND	ND	ND	ND	ND	ND
Benzo(g,h,i)perylene	ND	ND	4.21E-12	ND	ND	1.80E-11	1.80E-11
Beryllium	ND	ND	1.29E-11	ND	ND	5.50E-11	5.50E-11
Cadmium	ND	ND	ND	ND	ND	ND	ND
Chromium	ND	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	1.73E-09	ND	ND	ND	ND
Chrysene	1.14E-14	1.90E-14	7.98E-16	1.06E-15	3.31E-14	7.40E-09	7.40E-09
Dibenzof(a,h)anthracene	1.04E-11	8.56E-12	4.53E-13	9.63E-13	1.49E-11	3.41E-15	4.28E-14
Fluoranthene	ND	ND	ND	ND	ND	1.93E-12	4.47E-11
Fluorene	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	ND	ND	ND	ND	ND
Indeno(1,2,3-cd)pyrene	1.07E-12	4.52E-13	1.80E-10	ND	ND	7.68E-10	7.68E-10
Lead	ND	ND	5.80E-14	9.95E-14	7.86E-13	2.48E-13	6.34E-12
Naphthalene	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	2.85E-08	ND	ND	1.22E-07	1.22E-07
Phenanthrene	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND
Total Risk	1.10E-10	3.47E-08	3.07E-08	1.02E-11	6.05E-08	1.31E-07	1.58E-09

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-5

Summary of Potential Hazard Indices for Fuel Oil High Load Conditions
Fisher Residing at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	HI	Soil	Water	Air	Produce	Fish	HI
Mercury, elemental	NC	NC	4.16E-11	NC	NC	4.16E-11	NC	NC	3.55E-11	NC	NC	3.55E-11
Mercury, methyl	1.31E-05	9.33E-07	NC	2.39E-06	9.03E-02	9.03E-02	2.43E-07	3.25E-07	NC	1.42E-06	1.21E-01	1.21E-01
Mercury, divalent	7.36E-04	3.68E-06	2.72E-09	2.38E-05	NC	7.63E-04	1.37E-05	1.28E-06	2.32E-09	1.40E-05	NC	2.90E-05
1,4-Dichlorobenzene	2.00E-11	7.91E-10	8.86E-12	2.68E-08	3.20E-09	3.08E-08	3.71E-13	2.75E-10	7.57E-12	1.49E-08	4.27E-09	1.95E-08
Acenaphthene	2.42E-09	1.25E-09	9.96E-12	4.65E-08	1.08E-08	6.10E-08	4.49E-11	4.35E-10	8.51E-12	2.60E-08	1.44E-08	4.09E-08
Acenaphthylene	6.58E-08	2.12E-08	1.19E-13	3.07E-08	3.59E-06	3.71E-06	1.22E-09	7.38E-09	1.02E-13	1.84E-08	4.80E-06	4.82E-06
Anthracene	2.56E-10	1.69E-11	1.15E-13	2.26E-10	6.25E-10	1.12E-09	4.75E-12	5.88E-12	9.83E-14	1.35E-10	8.35E-10	9.81E-10
Arsenic	1.38E-04	8.18E-04	1.49E-08	6.51E-04	2.33E-04	1.84E-03	2.56E-06	2.85E-04	1.27E-08	3.90E-04	3.11E-04	9.89E-04
Benzene	9.26E-11	1.81E-07	3.95E-09	2.98E-07	6.37E-08	5.46E-07	1.72E-12	6.29E-08	3.37E-09	1.69E-07	8.51E-08	3.20E-07
Benzo(a)anthracene	1.25E-07	4.22E-09	7.10E-12	1.50E-08	3.06E-07	4.51E-07	2.33E-09	1.47E-09	6.07E-12	8.99E-09	4.09E-07	4.22E-07
Benzo(a)pyrene	2.77E-08	9.62E-10	1.95E-12	1.94E-08	1.36E-07	1.84E-07	5.15E-10	3.35E-10	1.66E-12	1.16E-08	1.82E-07	1.94E-07
Benzo(b)fluoranthene	9.06E-08	1.65E-09	5.42E-12	1.42E-08	2.33E-07	3.40E-07	1.68E-09	5.74E-10	4.63E-12	8.48E-09	3.12E-07	3.23E-07
Benzo(k)fluoranthene	2.87E-07	6.04E-09	5.42E-12	6.55E-08	8.55E-07	1.21E-06	5.33E-09	2.10E-09	4.63E-12	3.93E-08	1.14E-06	1.19E-06
Benzo(g,h,i)perylene	5.87E-07	1.89E-07	1.60E-11	6.24E-08	3.20E-05	3.28E-05	1.09E-08	6.58E-08	1.36E-11	3.72E-08	4.28E-05	4.29E-05
Beryllium	1.89E-05	4.89E-06	2.85E-08	3.58E-06	4.31E-06	3.17E-05	3.50E-07	1.70E-06	2.43E-08	2.15E-06	5.76E-06	9.99E-06
Cadmium	3.55E-06	8.26E-06	1.48E-10	1.47E-05	2.94E-05	5.59E-05	6.59E-08	2.88E-06	1.27E-10	8.80E-06	3.92E-05	5.10E-05
Chromium	3.16E-06	7.50E-10	4.87E-12	2.26E-07	2.03E-09	3.39E-06	5.87E-08	2.61E-10	4.16E-12	1.36E-07	2.71E-09	1.97E-07
Chromium VI	1.17E-05	1.06E-04	7.30E-08	8.26E-05	4.51E-06	2.04E-04	2.18E-07	3.68E-05	6.24E-08	4.96E-05	6.02E-06	9.26E-05
Chrysene	1.08E-07	5.13E-09	4.22E-12	1.64E-08	4.40E-07	5.70E-07	2.00E-09	1.79E-09	3.60E-12	9.81E-09	5.88E-07	6.02E-07
Dibenzo(a,h)anthracene	1.86E-07	2.28E-09	2.95E-12	9.98E-08	4.16E-07	7.04E-07	3.48E-09	7.95E-10	2.52E-12	5.99E-08	5.56E-07	6.20E-07
Fluoranthene	3.74E-08	1.69E-09	3.42E-12	3.68E-09	3.77E-07	4.20E-07	6.95E-10	5.87E-10	2.92E-12	2.17E-09	5.03E-07	5.07E-07
Fluorene	5.56E-09	2.38E-08	3.16E-12	4.65E-09	4.07E-07	4.41E-07	1.03E-10	8.30E-09	2.70E-12	2.79E-09	5.44E-07	5.55E-07
Formaldehyde	4.19E-11	2.98E-07	5.18E-09	1.35E-06	4.53E-09	1.65E-06	7.78E-13	1.04E-07	4.33E-12	7.65E-07	6.05E-09	8.79E-07
Indeno(1,2,3-cd)pyrene	1.87E-07	1.18E-09	3.78E-12	1.28E-07	2.21E-07	5.37E-07	3.48E-09	4.12E-10	3.23E-12	7.67E-08	2.95E-07	3.75E-07
Lead	9.98E-03	2.32E-03	1.67E-07	2.07E-03	2.64E-04	1.46E-02	1.85E-04	8.07E-04	1.43E-07	1.24E-03	3.52E-04	2.58E-03
Naphthalene	3.14E-08	1.52E-07	2.00E-09	4.77E-06	4.64E-07	5.42E-06	5.83E-10	5.28E-08	1.71E-09	2.69E-06	6.19E-07	3.36E-06
Nickel	2.19E-03	5.86E-03	3.70E-04	4.88E-03	6.50E-03	1.98E-02	4.06E-05	2.04E-03	3.16E-04	2.93E-03	8.68E-03	1.40E-02
Phenanthrene	2.55E-10	7.28E-09	7.42E-11	9.40E-03	3.42E-07	3.59E-07	4.73E-12	2.54E-09	6.34E-11	5.63E-09	4.57E-07	4.65E-07
Pyrene	1.47E-07	4.75E-08	4.01E-12	6.88E-08	8.04E-06	8.31E-06	2.74E-09	1.65E-08	3.42E-12	4.12E-08	1.07E-05	1.08E-05
Toluene	8.79E-11	7.53E-08	1.56E-10	2.03E-07	6.72E-08	3.45E-07	1.63E-12	2.62E-08	1.33E-10	1.14E-07	8.98E-08	2.30E-07
Vanadium	2.46E-02	7.72E-02	1.54E-06	6.80E-02	1.10E-02	1.81E-01	4.56E-04	2.69E-02	1.32E-06	4.08E-02	1.47E-02	8.28E-02
Total HI	3.77E-02	8.63E-02	3.72E-04	7.57E-02	1.08E-01	3.08E-01	6.99E-04	3.00E-02	3.18E-04	4.54E-02	1.45E-01	2.21E-01

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-6
Summary of Potential Excess Lifetime Cancer Risk for Fuel Oil High Load Conditions
Fisher Residing at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child					Adult				
	Soil	Water	Air	Produce	Risk	Soil	Water	Air	Produce	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	9.46E-15	3.74E-13	4.17E-15	1.26E-11	1.51E-12	8.79E-16	6.51E-13	1.78E-14	3.52E-11	1.01E-11
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	5.84E-09	3.47E-08	8.58E-12	2.77E-08	9.88E-09	5.42E-10	6.05E-08	3.67E-11	8.31E-08	2.10E-07
Benzene	6.90E-16	1.35E-12	1.67E-14	2.22E-12	4.75E-13	6.41E-17	2.34E-12	7.13E-14	6.29E-12	3.17E-12
Benzo(a)anthracene	4.54E-11	1.61E-12	3.02E-15	5.79E-12	1.17E-10	4.21E-12	2.79E-12	1.29E-14	1.73E-11	7.78E-10
Benzo(a)pyrene	1.02E-10	3.71E-12	8.28E-15	7.62E-11	5.26E-10	9.46E-12	6.47E-12	3.54E-14	2.29E-10	3.51E-09
Benzo(b)fluoranthene	3.29E-11	6.33E-13	2.30E-15	5.54E-12	8.96E-11	3.06E-12	1.10E-12	9.84E-15	1.66E-11	3.76E-09
Benzo(k)fluoranthene	8.59E-12	2.03E-13	2.30E-16	2.54E-12	2.87E-11	7.98E-13	3.53E-13	9.84E-16	7.61E-12	5.98E-10
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.92E-10
Beryllium	ND	ND	1.17E-13	ND	ND	ND	ND	ND	ND	ND
Cadmium	ND	ND	3.58E-13	ND	ND	ND	ND	4.99E-13	ND	4.99E-13
Chromium	ND	ND	ND	ND	ND	ND	ND	1.53E-12	ND	1.53E-12
Chromium VI	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chrysene	3.74E-13	1.90E-14	1.79E-17	6.30E-14	1.63E-12	ND	ND	2.06E-10	ND	2.06E-10
Dibenz(a,h)anthracene	6.45E-10	8.56E-12	1.25E-14	3.93E-10	2.09E-12	3.48E-14	3.31E-14	7.66E-17	1.89E-13	1.09E-11
Fluoranthene	ND	ND	ND	ND	ND	5.99E-11	1.49E-11	5.36E-14	1.18E-09	1.04E-08
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Indeno(1,2,3-cd)pyrene	6.68E-11	4.52E-13	1.61E-15	5.04E-11	8.42E-11	6.20E-12	7.86E-13	6.86E-15	1.51E-10	5.62E-10
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	7.91E-10	ND	ND	ND	ND	ND	ND	ND
Phenanthrene	ND	ND	ND	ND	7.91E-10	ND	ND	3.38E-09	ND	3.38E-09
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total Risk	6.74E-09	3.47E-08	8.52E-10	2.82E-08	1.23E-08	6.25E-10	6.05E-08	3.64E-09	8.47E-08	8.21E-08

Notes: ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-7
Summary of Potential Hazard Indices for Fuel Oil High Load Conditions
Fisher Residing at Saint John
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	HI	Soil	Water	Air	Produce	Fish	HI
Mercury, elemental	NC	NC	1.87E-09	NC	NC	1.87E-09	NC	NC	1.60E-09	NC	NC	1.60E-09
Mercury, methyl	4.14E-07	9.33E-07	NC	6.20E-08	9.03E-02	9.03E-02	7.69E-09	3.25E-07	NC	3.67E-08	1.21E-01	1.60E-09
Mercury, divalent	2.32E-05	3.68E-06	1.22E-07	8.26E-07	NC	2.79E-05	4.32E-07	1.28E-06	1.04E-07	4.87E-07	NC	2.30E-06
1,4-Dichlorobenzene	6.66E-13	7.91E-10	3.99E-10	8.91E-10	3.20E-09	5.28E-09	1.24E-14	2.75E-10	3.41E-10	4.97E-10	4.27E-09	5.38E-09
Acenaphthene	8.06E-11	1.25E-09	4.48E-10	1.55E-09	1.08E-08	1.41E-08	1.50E-12	4.35E-10	3.83E-10	8.68E-10	1.44E-08	1.61E-08
Acenaphthylene	1.56E-09	2.12E-08	5.17E-12	8.39E-10	3.59E-06	3.61E-06	2.91E-11	7.38E-09	4.42E-12	5.03E-10	4.80E-06	4.80E-06
Anthracene	8.51E-12	1.69E-11	5.18E-12	7.53E-12	6.25E-10	6.63E-10	1.58E-13	5.88E-12	4.42E-12	4.50E-12	8.35E-10	8.50E-10
Arsenic	2.20E-06	8.18E-04	5.36E-07	1.21E-05	2.33E-04	1.07E-03	4.09E-08	2.85E-04	4.57E-07	7.23E-06	3.11E-04	6.04E-04
Benzene	3.08E-12	1.81E-07	1.78E-07	9.91E-09	6.37E-08	4.32E-07	5.73E-14	6.29E-08	1.52E-07	5.62E-09	8.51E-08	3.05E-07
Benzof(a)anthracene	4.01E-09	4.22E-09	3.18E-10	4.10E-10	3.06E-07	3.15E-07	7.44E-11	1.47E-09	2.71E-10	2.45E-10	4.09E-07	4.11E-07
Benzof(a)pyrene	6.83E-10	9.62E-10	8.48E-11	4.77E-10	1.36E-07	1.38E-07	1.27E-11	3.35E-10	7.24E-11	2.86E-10	1.82E-07	1.83E-07
Benzof(b)fluoranthene	2.83E-09	1.65E-09	2.42E-10	3.82E-10	2.33E-07	2.39E-07	5.26E-11	5.74E-10	2.07E-10	2.29E-10	3.12E-07	3.13E-07
Benzof(k)fluoranthene	6.65E-09	6.04E-09	2.35E-10	1.63E-09	8.55E-07	8.70E-07	1.24E-10	2.10E-09	2.00E-10	9.75E-10	1.14E-06	1.15E-06
Benzof(g,h,i)perylene	1.40E-08	1.89E-07	6.92E-10	3.50E-09	3.20E-05	3.22E-05	2.59E-10	6.58E-08	5.91E-10	2.09E-09	4.28E-05	4.28E-05
Beryllium	3.02E-07	4.89E-06	1.02E-06	6.63E-08	4.31E-06	1.06E-05	5.60E-09	1.70E-06	8.76E-07	3.98E-08	5.76E-06	8.39E-06
Cadmium	5.67E-08	8.26E-06	5.34E-09	2.52E-07	2.94E-05	3.79E-05	1.05E-09	2.88E-06	4.56E-09	1.51E-07	3.92E-05	4.23E-05
Chromium	5.06E-08	7.50E-10	1.75E-10	4.16E-09	2.03E-09	5.77E-08	9.40E-10	2.61E-10	1.50E-10	2.49E-09	2.71E-09	6.55E-09
Chromium VI	1.87E-07	1.08E-04	2.63E-06	1.53E-06	4.51E-06	1.14E-04	3.48E-09	3.68E-05	2.25E-06	9.21E-07	6.02E-06	4.59E-05
Chrysene	3.30E-09	5.13E-09	1.88E-10	4.54E-10	4.40E-07	4.49E-07	6.12E-11	1.79E-09	1.60E-10	2.71E-10	5.88E-07	5.90E-07
Dibenzof(a,h)anthracene	2.99E-09	2.28E-09	1.07E-10	2.28E-09	4.16E-07	4.24E-07	5.56E-11	7.95E-10	9.10E-11	1.37E-09	5.56E-07	5.58E-07
Fluoranthene	1.24E-09	1.69E-09	1.54E-10	1.19E-10	3.77E-07	3.80E-07	2.31E-11	5.87E-10	1.31E-10	7.05E-11	5.03E-07	5.04E-07
Fluorene	1.81E-10	2.38E-08	1.42E-10	1.36E-10	4.07E-07	4.31E-07	3.36E-12	8.30E-09	1.21E-10	8.13E-11	5.44E-07	5.52E-07
Formaldehyde	1.39E-12	2.98E-07	2.33E-07	4.48E-08	4.53E-09	5.80E-07	2.59E-14	1.04E-07	1.99E-07	2.55E-08	6.05E-09	3.34E-07
Indeno(1,2,3-cd)pyrene	3.00E-09	1.18E-09	1.36E-10	4.40E-09	2.21E-07	2.29E-07	5.58E-11	4.12E-10	1.16E-10	2.64E-09	2.95E-07	2.98E-07
Lead	1.60E-04	2.32E-03	6.02E-06	3.73E-05	2.64E-04	2.78E-03	2.97E-06	8.07E-04	5.14E-06	2.24E-05	3.52E-04	1.19E-03
Naphthalene	1.05E-09	1.52E-07	8.99E-08	1.59E-07	4.64E-07	8.65E-07	1.94E-11	5.28E-08	7.68E-08	8.95E-08	6.19E-07	8.38E-07
Nickel	3.50E-05	5.86E-03	1.33E-02	8.97E-05	6.50E-03	2.58E-02	6.49E-07	2.04E-03	1.14E-02	5.38E-05	8.68E-03	2.22E-02
Phenanthrene	8.48E-12	7.28E-09	3.34E-09	3.13E-10	3.42E-07	3.53E-07	1.57E-13	2.54E-09	2.85E-09	1.87E-10	4.57E-07	4.62E-07
Pyrene	3.51E-09	4.75E-08	1.74E-10	1.88E-09	8.04E-06	8.10E-06	6.51E-11	1.65E-08	1.49E-10	1.13E-09	1.07E-05	1.08E-05
Toluene	2.93E-12	7.53E-08	7.01E-09	6.75E-09	6.72E-08	1.56E-07	5.44E-14	2.62E-08	5.99E-09	3.79E-09	8.98E-08	1.26E-07
Vanadium	3.93E-04	7.72E-02	5.55E-05	1.26E-03	1.10E-02	8.99E-02	7.30E-06	2.69E-02	4.74E-05	7.54E-04	1.47E-02	4.24E-02
Total HI	6.14E-04	8.63E-02	1.34E-02	1.40E-03	1.08E-01	2.10E-01	1.14E-05	3.00E-02	1.14E-02	8.39E-04	1.45E-01	1.87E-01

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-8
Summary of Potential Excess Lifetime Cancer Risk for Fuel Oil High Load Conditions
Fisher Residing at Saint John
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child					Adult						
	Soil	Water	Air	Produce	Fish	Risk	Soil	Water	Air	Produce	Fish	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	3.15E-16	3.74E-13	1.87E-13	4.21E-13	1.51E-12	2.49E-12	2.93E-17	6.51E-13	8.01E-13	1.17E-12	1.01E-11	1.27E-11
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	9.33E-11	3.47E-08	3.09E-10	5.14E-10	9.88E-09	4.55E-08	8.67E-12	6.05E-08	1.32E-09	1.54E-09	6.60E-08	1.29E-07
Benzene	2.30E-17	1.35E-12	7.51E-13	7.39E-14	4.75E-13	2.65E-12	2.13E-18	2.34E-12	3.21E-12	2.10E-13	3.17E-12	8.93E-12
Benzo(a)anthracene	1.45E-12	1.61E-12	1.35E-13	1.57E-13	1.17E-10	1.20E-10	1.35E-13	2.79E-12	5.77E-13	4.70E-13	7.78E-10	7.82E-10
Benzo(a)pyrene	2.51E-12	3.71E-12	3.60E-13	1.88E-12	5.26E-10	5.34E-10	2.33E-13	6.47E-12	1.54E-12	5.64E-12	3.51E-09	3.53E-09
Benzo(b)fluoranthene	1.03E-12	6.33E-13	1.03E-13	1.49E-13	8.96E-11	9.15E-11	9.56E-14	1.10E-12	4.39E-13	4.47E-13	5.98E-10	6.00E-10
Benzo(k)fluoranthene	1.99E-13	2.03E-13	9.97E-15	6.31E-14	2.87E-11	2.92E-11	1.85E-14	3.53E-13	4.26E-14	1.89E-13	1.92E-10	1.92E-10
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	ND	ND	4.21E-12	ND	ND	4.21E-12	ND	ND	1.80E-11	ND	ND	1.80E-11
Cadmium	ND	ND	1.29E-11	ND	ND	1.29E-11	ND	ND	5.50E-11	ND	ND	5.50E-11
Chromium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	1.73E-09	ND	ND	1.73E-09	ND	ND	7.40E-09	ND	ND	7.40E-09
Chrysene	1.14E-14	1.90E-14	7.98E-16	1.74E-15	1.63E-12	1.66E-12	1.06E-15	3.31E-14	3.41E-15	5.21E-15	1.09E-11	1.09E-11
Dibenz(a,h)anthracene	1.04E-11	8.56E-12	4.53E-13	8.97E-12	1.56E-09	1.59E-09	9.63E-13	1.49E-11	1.93E-12	2.69E-11	1.04E-08	1.05E-08
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	1.80E-10	ND	ND	1.80E-10	ND	ND	7.68E-10	ND	ND	7.68E-10
Indeno(1,2,3-cd)pyrene	1.07E-12	4.52E-13	5.80E-14	1.73E-12	8.42E-11	8.75E-11	9.95E-14	7.86E-13	2.48E-13	5.20E-12	5.62E-10	5.69E-10
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	2.85E-08	ND	ND	2.85E-08	ND	ND	1.22E-07	ND	ND	1.22E-07
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total Risk	1.10E-10	3.47E-08	3.07E-08	5.27E-10	1.23E-08	7.84E-08	1.02E-11	6.05E-08	1.31E-07	1.58E-09	8.21E-08	2.75E-07

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-9
Summary of Potential Hazard Indices for Fuel Oil High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child									
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	HI
Mercury, elemental	NC	NC	9.24E-10	NC	NC	1.13E-07	5.96E-10	6.14E-10	NC	9.24E-10
Mercury, methyl	2.37E-07	9.33E-07	NC	1.44E-07	6.35E-09	1.36E-13	1.36E-13	1.36E-13	1.36E-13	1.36E-13
Mercury, divalent	1.33E-05	3.68E-06	6.01E-08	1.86E-06	1.40E-06	1.88E-05	2.23E-07	2.30E-07	1.90E-11	1.31E-06
1,4-Dichlorobenzene	3.80E-13	7.91E-10	1.97E-10	2.03E-09	2.33E-14	6.07E-13	1.14E-15	4.72E-13	2.93E-14	3.02E-09
Acenaphthene	4.60E-11	1.25E-09	2.21E-10	3.54E-09	1.08E-12	1.91E-11	1.34E-14	5.52E-12	3.94E-13	5.05E-09
Acenaphthylene	9.22E-10	2.12E-08	2.56E-12	1.92E-09	4.85E-10	8.25E-09	1.33E-12	5.52E-10	5.58E-11	2.40E-08
Anthracene	4.86E-12	1.69E-11	2.56E-12	1.72E-11	2.07E-13	3.22E-12	2.64E-15	1.09E-12	7.02E-14	4.15E-11
Arsenic	1.22E-06	8.18E-04	2.68E-07	2.58E-05	2.25E-06	3.74E-06	NC	NC	NC	8.45E-04
Benzene	1.76E-12	1.81E-07	8.77E-08	2.26E-08	3.52E-14	9.36E-13	1.36E-15	5.60E-13	4.57E-14	2.91E-07
Benzo(a)anthracene	2.29E-09	4.22E-09	1.57E-10	9.42E-10	1.32E-09	1.90E-08	1.54E-11	6.35E-09	4.04E-10	7.61E-09
Benzo(a)pyrene	4.01E-10	9.62E-10	4.20E-11	1.11E-09	2.69E-09	4.55E-08	7.55E-12	3.12E-09	3.02E-10	2.51E-09
Benzo(b)fluoranthene	1.63E-09	1.65E-09	1.20E-10	8.77E-10	4.13E-09	6.30E-08	3.62E-11	1.50E-08	1.02E-09	4.27E-09
Benzo(k)fluoranthene	3.93E-09	6.04E-09	1.16E-10	3.77E-09	1.31E-08	2.07E-07	8.70E-11	3.60E-08	2.60E-09	1.39E-08
Benzo(g,h,i)perylene	8.22E-09	1.89E-07	3.43E-10	7.29E-09	3.26E-09	5.46E-08	1.19E-11	4.92E-09	4.54E-10	2.05E-07
Beryllium	1.66E-07	4.89E-06	5.12E-07	1.42E-07	9.66E-09	4.11E-10	NC	NC	NC	5.71E-06
Cadmium	3.13E-08	8.26E-06	2.67E-09	5.48E-07	1.94E-09	5.97E-09	8.04E-09	1.96E-10	3.62E-10	8.84E-06
Chromium	2.79E-08	7.50E-10	8.76E-11	8.92E-09	5.27E-09	5.93E-08	NC	NC	NC	3.77E-08
Chromium VI	1.03E-07	1.06E-04	1.31E-06	3.29E-06	7.81E-07	1.18E-05	NC	NC	NC	1.10E-04
Chrysene	1.89E-09	5.13E-09	9.28E-11	1.04E-09	1.69E-08	2.58E-08	1.46E-11	6.01E-09	4.11E-10	8.16E-09
Dibenz(a,h)anthracene	1.65E-09	2.28E-09	5.32E-11	4.81E-09	4.84E-08	8.34E-07	8.13E-11	3.35E-08	4.58E-09	8.80E-09
Fluoranthene	7.10E-10	1.69E-09	7.60E-11	2.71E-10	8.24E-11	1.12E-09	1.23E-12	5.09E-10	3.05E-11	2.74E-09
Fluorene	1.03E-10	2.38E-08	7.01E-11	3.09E-10	1.07E-11	1.84E-10	2.29E-14	9.47E-12	1.02E-12	2.43E-08
Formaldehyde	7.96E-13	2.98E-07	1.15E-07	1.02E-07	2.78E-15	7.39E-14	1.07E-16	4.41E-14	3.60E-15	5.15E-07
Indeno(1,2,3-cd)pyrene	1.66E-09	1.18E-09	6.82E-11	9.09E-09	3.90E-07	6.84E-06	1.90E-10	7.63E-06	2.86E-08	1.20E-08
Lead	8.80E-05	2.32E-03	3.01E-06	8.04E-05	1.77E-06	7.16E-05	NC	NC	4.08E-07	2.49E-03
Naphthalene	5.97E-10	1.52E-07	4.44E-08	3.63E-07	1.19E-11	2.65E-10	2.85E-13	1.18E-10	9.44E-12	5.59E-07
Nickel	1.93E-05	5.86E-03	6.66E-03	1.93E-04	4.97E-05	4.55E-04	NC	NC	NC	1.27E-02
Phenanthrene	4.84E-12	7.28E-09	1.65E-09	7.15E-10	9.62E-13	2.40E-11	3.55E-14	1.46E-11	1.17E-12	9.65E-09
Pyrene	2.07E-09	4.75E-08	8.62E-11	4.30E-09	1.09E-09	1.85E-08	2.99E-12	1.24E-09	1.25E-10	5.39E-08
Toluene	1.67E-12	7.53E-08	3.46E-09	1.54E-08	5.42E-14	1.43E-12	2.07E-15	8.53E-13	6.95E-14	9.42E-08
Vanadium	2.17E-04	7.72E-02	2.77E-05	2.70E-03	2.91E-04	1.28E-02	NC	NC	NC	8.01E-02
Total HI	3.39E-04	8.63E-02	6.70E-03	3.00E-03	3.48E-04	1.34E-02	2.32E-07	4.17E-07	4.52E-07	9.63E-02

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-9
Summary of Potential Hazard Indices for Fuel Oil High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Adult									
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	HI
Mercury, elemental	NC	NC	7.89E-10	NC	NC	NC	NC	NC	NC	7.89E-10
Mercury, methyl	4.41E-09	3.25E-07	NC	8.52E-08	1.23E-08	4.99E-08	7.41E-10	7.54E-10	1.61E-11	4.14E-07
Mercury, divalent	2.47E-07	1.28E-06	5.13E-08	1.10E-06	2.72E-06	8.26E-06	2.78E-07	2.82E-07	5.78E-09	2.68E-06
1,4-Dichlorobenzene	7.06E-15	2.75E-10	1.68E-10	1.13E-09	4.52E-14	2.67E-13	1.42E-15	5.79E-13	3.39E-14	1.59E-09
Acenaphthene	8.54E-13	4.35E-10	1.89E-10	1.98E-09	2.10E-12	8.41E-12	1.67E-14	6.77E-12	4.55E-13	2.61E-09
Acenaphthylene	1.71E-11	7.38E-09	2.19E-12	1.15E-09	9.40E-10	3.63E-09	1.66E-12	6.77E-10	6.44E-11	8.55E-09
Anthracene	9.02E-14	5.88E-12	2.18E-12	1.03E-11	4.02E-13	1.42E-12	3.29E-15	1.34E-12	8.10E-14	1.84E-11
Arsenic	2.26E-08	2.85E-04	2.29E-07	1.55E-05	4.35E-06	1.64E-06	NC	NC	NC	3.01E-04
Benzene	3.27E-14	6.29E-08	7.50E-08	1.28E-08	6.82E-14	4.12E-13	1.69E-15	6.87E-13	5.28E-14	1.51E-07
Benzo(a)anthracene	4.26E-11	1.47E-09	1.34E-10	5.63E-10	2.56E-09	8.36E-09	1.91E-11	7.78E-09	4.67E-10	2.21E-09
Benzo(a)pyrene	7.45E-12	3.35E-10	3.59E-11	6.63E-10	5.21E-09	2.00E-08	9.39E-12	3.82E-09	3.49E-10	1.04E-09
Benzo(b)fluoranthene	3.02E-11	5.74E-10	1.02E-10	5.25E-10	8.00E-09	2.77E-08	4.50E-11	1.84E-08	1.18E-09	1.23E-09
Benzo(k)fluoranthene	7.30E-11	2.10E-09	9.93E-11	2.26E-09	2.54E-08	9.12E-08	1.08E-10	4.41E-08	3.00E-09	4.53E-09
Benzo(g,h,i)perylene	1.53E-10	6.58E-08	2.93E-10	4.36E-09	6.31E-09	2.40E-08	1.48E-11	6.03E-09	5.23E-10	7.06E-08
Beryllium	3.09E-09	1.70E-06	4.38E-07	8.53E-08	1.87E-08	1.81E-10	NC	NC	NC	2.23E-06
Cadmium	5.81E-10	2.88E-06	2.28E-09	3.27E-07	3.76E-09	2.62E-09	1.00E-08	2.40E-10	4.18E-10	3.21E-06
Chromium	5.18E-10	2.61E-10	7.49E-11	5.35E-09	1.02E-08	2.61E-08	NC	NC	NC	6.20E-09
Chromium VI	1.92E-09	3.68E-05	1.12E-06	1.97E-06	1.51E-06	5.20E-06	NC	NC	NC	3.99E-05
Chrysene	3.52E-11	1.79E-09	7.93E-11	6.21E-10	3.28E-09	1.14E-08	1.82E-11	7.38E-09	4.74E-10	2.52E-09
Dibenzo(a,h)anthracene	3.07E-11	7.95E-10	4.55E-11	2.89E-09	9.38E-08	3.67E-07	1.01E-10	4.11E-08	5.26E-09	3.76E-09
Fluoranthene	1.32E-11	5.87E-10	6.49E-11	1.61E-10	1.60E-10	4.91E-10	1.53E-12	6.24E-10	3.52E-11	8.26E-10
Fluorene	1.92E-12	8.30E-09	5.99E-11	1.85E-10	2.08E-11	8.08E-11	2.85E-14	1.16E-11	1.18E-12	8.55E-09
Formaldehyde	1.48E-14	1.04E-07	9.84E-08	5.82E-08	5.38E-15	3.25E-14	1.33E-16	5.41E-14	4.16E-15	2.60E-07
Indeno(1,2,3-cd)pyrene	3.08E-11	4.12E-10	5.82E-11	5.45E-09	7.55E-07	3.01E-06	2.36E-10	9.61E-08	3.30E-08	5.95E-09
Lead	1.63E-06	8.07E-04	2.57E-06	4.81E-05	3.42E-06	3.15E-05	NC	NC	4.71E-07	8.59E-04
Naphthalene	1.11E-11	5.28E-08	3.79E-08	2.04E-07	2.30E-11	1.17E-10	3.55E-13	1.44E-10	1.09E-11	2.95E-07
Nickel	3.58E-07	2.04E-03	5.69E-03	1.15E-04	9.64E-05	2.00E-04	NC	NC	NC	7.85E-03
Phenanthrene	8.99E-14	2.54E-09	1.41E-09	4.28E-10	1.86E-12	1.05E-11	4.41E-14	1.80E-11	1.36E-12	4.37E-09
Pyrene	3.84E-11	1.65E-08	7.36E-11	2.58E-09	2.11E-09	8.12E-09	3.72E-12	1.52E-09	1.44E-10	1.92E-08
Toluene	3.10E-14	2.62E-08	2.96E-09	8.66E-09	1.05E-13	6.28E-13	2.57E-15	1.05E-12	8.02E-14	3.78E-08
Vanadium	4.02E-06	2.69E-02	2.37E-05	1.62E-03	5.64E-04	5.63E-03	NC	NC	NC	2.85E-02
Total HI	6.30E-06	3.00E-02	5.72E-03	1.80E-03	6.74E-04	5.89E-03	2.89E-07	5.11E-07	5.22E-07	3.76E-02

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-10
Summary of Potential Excess Lifetime Cancer Risk for Fuel Oil High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Loneville, New Brunswick

Compound	Child										Risk
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork		
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
1,4-Dichlorobenzene	1.80E-16	3.74E-13	9.26E-14	9.60E-13	1.1E-17	2.863E-16	5.402E-19	2.228E-16	1.384E-17	1.43E-12	
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Arsenic	5.15E-11	3.47E-08	1.54E-10	1.10E-09	9.628E-11	1.602E-10	NC	NC	NC	3.60E-08	
Benzene	1.31E-17	1.35E-12	3.71E-13	1.69E-13	2.624E-19	6.98E-18	1.011E-20	4.176E-18	3.409E-19	1.89E-12	
Benzo(a)anthracene	8.30E-13	1.61E-12	6.68E-14	3.62E-13	4.984E-13	7.228E-12	5.561E-15	2.296E-12	1.474E-13	2.86E-12	
Benzo(a)pyrene	1.47E-12	3.71E-12	1.79E-13	4.35E-12	1.051E-11	1.783E-10	2.77E-14	1.144E-11	1.753E-12	9.72E-12	
Benzo(b)fluoranthene	5.91E-13	6.33E-13	5.08E-14	3.42E-13	1.583E-12	2.437E-11	1.315E-14	5.447E-12	3.753E-13	1.62E-12	
Benzo(k)fluoranthene	1.18E-13	2.03E-13	4.94E-15	1.46E-13	4.832E-13	7.818E-12	2.604E-15	1.077E-12	8.238E-14	4.71E-13	
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Beryllium	ND	ND	2.10E-12	ND	ND	ND	ND	ND	ND	2.10E-12	
Cadmium	ND	ND	6.44E-12	ND	ND	ND	ND	ND	ND	6.44E-12	
Chromium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Chromium VI	ND	ND	8.66E-10	ND	ND	ND	ND	ND	ND	8.66E-10	
Chrysene	6.58E-15	1.90E-14	3.95E-16	3.98E-15	6.358E-15	9.82E-14	5.074E-17	2.087E-14	1.454E-15	3.00E-14	
Dibenzo(a,h)anthracene	5.72E-12	8.56E-12	2.26E-13	1.89E-11	1.894E-10	3.275E-09	2.814E-13	1.16E-10	1.704E-11	3.34E-11	
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Formaldehyde	ND	ND	8.88E-11	ND	ND	ND	ND	ND	ND	8.88E-11	
Indeno(1,2,3-cd)pyrene	5.91E-13	4.52E-13	2.90E-14	3.58E-12	1.534E-10	2.695E-09	6.764E-14	2.793E-11	1.11E-11	4.65E-12	
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Nickel	ND	ND	1.42E-08	ND	ND	ND	ND	ND	ND	ND	
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.42E-08	
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Total Risk	6.08E-11	3.47E-08	1.54E-08	1.13E-09	4.52E-10	6.35E-09	3.98E-13	1.64E-10	2.99E-11	5.13E-08	

Notes:
ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-10

Summary of Potential Excess Lifetime Cancer Risk for Fuel Oil High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Adult									
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	1.67E-17	6.51E-13	3.95E-13	2.68E-12	1.066E-16	6.293E-16	3.36E-18	1.367E-15	7.988E-17	3.72E-12
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	4.78E-12	6.05E-08	6.60E-10	3.30E-09	9.326E-10	3.521E-10	NC	NC	NC	6.44E-08
Benzene	1.22E-18	2.34E-12	1.58E-12	4.79E-13	2.542E-18	1.534E-17	6.289E-20	2.562E-17	1.967E-18	4.41E-12
Benzo(a)anthracene	7.71E-14	2.79E-12	2.85E-13	1.08E-12	4.827E-12	1.589E-11	3.459E-14	1.408E-11	8.505E-13	4.24E-12
Benzo(a)pyrene	1.37E-13	6.47E-12	7.63E-13	1.31E-11	1.018E-10	3.921E-10	1.723E-13	7.018E-11	6.595E-12	2.04E-11
Benzo(b)fluoranthene	5.49E-14	1.10E-12	2.17E-13	1.03E-12	1.534E-11	5.358E-11	8.18E-14	3.341E-11	2.166E-12	2.40E-12
Benzo(k)fluoranthene	1.09E-14	3.53E-13	2.11E-14	4.38E-13	4.681E-12	1.719E-11	1.619E-14	6.605E-12	4.754E-13	8.22E-13
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	ND	ND	8.98E-12	ND	ND	ND	ND	ND	ND	8.98E-12
Cadmium	ND	ND	2.75E-11	ND	ND	ND	ND	ND	ND	2.75E-11
Chromium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	3.70E-09	ND	ND	ND	ND	ND	ND	ND
Chrysene	6.11E-16	3.31E-14	1.69E-15	1.19E-14	6.159E-14	2.159E-13	3.156E-16	1.28E-13	8.388E-15	3.70E-09
Dibenzo(a,h)anthracene	5.31E-13	1.49E-11	9.67E-13	5.68E-11	1.835E-09	7.199E-09	1.75E-12	7.117E-10	9.83E-11	4.73E-14
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	7.32E-11
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	3.79E-10	ND	ND	ND	ND	ND	ND	ND
Indeno(1,2,3-cd)pyrene	5.48E-14	7.86E-13	1.24E-13	1.07E-11	1.486E-09	5.925E-09	4.207E-13	1.713E-10	6.403E-11	3.79E-10
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.17E-11
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	6.08E-08	ND	ND	ND	ND	ND	ND	ND
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	6.08E-08
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total Risk	5.64E-12	6.05E-08	6.56E-08	3.39E-09	4.38E-09	1.40E-08	2.48E-12	1.01E-09	1.72E-10	1.29E-07

Notes:

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-11
Summary of Potential Hazard Indices for Orimulsion High Load Conditions
Resident at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Orneville, New Brunswick

Compound	Child				Adult			
	Soil	Water	Air	HI	Soil	Water	Air	HI
Mercury, elemental	2.45E-06	1.63E-07	6.11E-12	6.11E-12	NC	NC	5.22E-12	5.22E-12
	1.37E-04	6.36E-07	NC	3.06E-06	4.55E-08	5.66E-08	NC	2.65E-07
Mercury, methyl	1.37E-04	6.36E-07	4.00E-10	4.46E-06	4.55E-06	2.21E-07	3.42E-10	2.62E-06
	8.37E-12	2.62E-10	2.92E-12	1.12E-08	1.55E-13	9.13E-11	2.49E-12	6.34E-09
1,4-Dichlorobenzene	9.40E-10	3.98E-10	3.05E-12	1.81E-08	1.75E-11	1.39E-10	2.60E-12	1.01E-08
	2.57E-08	7.13E-09	3.66E-14	1.20E-08	4.49E-08	2.48E-09	3.12E-14	7.20E-09
Acenaphthylene	9.95E-11	5.55E-12	3.53E-14	8.81E-11	1.93E-10	1.93E-12	3.01E-14	5.65E-11
	1.28E-05	6.66E-05	1.07E-09	6.04E-05	1.40E-04	2.37E-07	2.32E-05	3.62E-05
Anthracene	1.38E-10	2.12E-07	4.62E-09	4.42E-07	2.55E-12	7.38E-08	3.95E-09	2.51E-07
	4.88E-08	1.45E-09	2.17E-12	5.86E-09	9.06E-10	5.06E-10	1.86E-12	3.51E-09
Benzene	1.17E-08	3.40E-10	6.43E-13	8.15E-09	2.02E-08	1.18E-10	5.49E-13	4.89E-09
	3.80E-08	6.00E-10	1.79E-12	5.96E-09	4.46E-08	7.06E-10	1.53E-12	3.57E-09
Benzol(a)fluoranthene	1.21E-07	2.16E-09	1.79E-12	2.76E-08	1.51E-07	7.52E-10	1.53E-12	1.65E-08
	2.30E-07	6.38E-08	4.91E-12	2.44E-08	3.18E-07	2.22E-08	4.19E-12	1.46E-08
Benzol(g,h,i)perylene	2.76E-06	6.31E-07	3.25E-09	5.25E-07	5.13E-08	2.20E-07	2.77E-09	3.15E-07
	3.31E-07	6.78E-07	1.08E-11	1.38E-06	2.38E-06	6.16E-09	9.22E-12	8.22E-07
Cadmium	4.25E-07	8.87E-11	5.09E-13	3.04E-08	4.55E-07	3.09E-11	4.35E-13	1.82E-08
	1.58E-06	1.25E-05	7.64E-09	1.11E-05	2.52E-05	2.93E-08	6.53E-09	6.67E-06
Chromium VI	4.19E-08	1.78E-09	1.29E-12	6.39E-09	5.01E-08	6.19E-10	1.10E-12	3.83E-09
	7.35E-08	7.76E-10	9.06E-13	3.94E-08	1.14E-07	2.70E-10	7.74E-13	2.36E-08
Dibenzol(a,h)anthracene	1.46E-08	5.91E-10	1.05E-12	1.43E-09	1.66E-08	2.06E-10	8.98E-13	8.48E-10
	2.16E-09	8.35E-09	9.68E-13	1.81E-09	1.23E-08	2.91E-09	8.27E-13	1.09E-09
Fluorene	1.15E-12	6.49E-09	1.12E-10	3.69E-08	4.35E-08	2.26E-09	9.56E-11	2.10E-08
	7.39E-08	4.01E-10	1.16E-12	5.05E-08	1.25E-07	1.37E-09	9.92E-13	3.03E-08
Formaldehyde	4.88E-06	9.98E-07	6.36E-11	1.01E-06	6.89E-06	9.06E-08	5.44E-11	6.05E-07
	4.16E-09	1.61E-08	2.09E-10	6.32E-07	6.53E-07	5.60E-09	1.78E-10	3.56E-07
Lead	3.88E-04	9.13E-04	5.11E-05	8.66E-04	2.22E-03	3.18E-04	4.37E-05	8.88E-04
	9.92E-11	2.24E-09	2.28E-11	3.66E-09	6.02E-09	1.84E-12	1.95E-11	2.19E-09
Naphthalene	5.76E-08	1.60E-08	1.23E-12	2.69E-08	1.07E-09	5.57E-09	1.05E-12	1.61E-08
	5.73E-11	3.88E-08	8.00E-11	1.32E-07	1.71E-07	1.06E-12	6.83E-11	7.42E-08
Phenanthrene	2.88E-03	7.95E-03	1.41E-07	7.97E-03	1.88E-02	5.35E-05	1.20E-07	4.78E-03
	3.43E-03	8.87E-03	5.13E-05	8.91E-03	2.13E-02	6.37E-05	4.38E-05	5.35E-03
Pyrene								
Toluene								
Vanadium								
Total HI								

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-12
Summary of Potential Excess Lifetime Cancer Risk for Orimulsion High Load Conditions
Resident at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child			Adult		
	Soil	Water	Air	Soil	Water	Air
Mercury, elemental	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	3.96E-15	1.24E-13	1.37E-15	3.68E-16	2.16E-13	5.87E-15
Acenaphthene	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND
Arsenic	5.41E-10	2.83E-09	6.19E-13	5.02E-11	4.92E-09	2.64E-12
Benzene	1.03E-15	1.58E-12	1.95E-14	9.52E-17	2.75E-12	9.35E-14
Benzo(a)anthracene	1.78E-11	5.51E-13	9.24E-16	1.64E-12	9.59E-13	3.95E-15
Benzo(a)pyrene	4.29E-11	1.31E-12	2.73E-15	3.98E-12	2.28E-12	1.17E-14
Benzo(b)fluoranthene	1.38E-11	2.30E-13	7.60E-16	1.28E-12	4.00E-13	3.25E-15
Benzo(k)fluoranthene	3.62E-12	7.22E-14	7.60E-17	3.36E-13	1.26E-13	3.25E-16
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND
Beryllium	ND	ND	1.33E-14	ND	ND	5.69E-14
Cadmium	ND	ND	2.60E-14	ND	ND	1.11E-13
Chromium	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	5.04E-12	ND	ND	2.15E-11
Chrysene	1.48E-13	6.56E-15	5.49E-18	1.35E-14	1.14E-14	2.34E-17
Dibenz(a,h)anthracene	2.54E-10	2.90E-12	3.85E-15	2.36E-11	5.05E-12	1.65E-14
Fluoranthene	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	8.63E-14	ND	ND	3.69E-13
Indeno(1,2,3-cd)pyrene	2.64E-11	1.53E-13	4.94E-16	2.45E-12	2.66E-13	2.11E-15
Lead	ND	ND	ND	ND	ND	ND
Naphthalene	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	1.09E-10	ND	ND	4.67E-10
Phenanthrene	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND
Total Risk	9.00E-10	4.67E-12	1.15E-10	8.36E-11	4.93E-09	4.92E-10
						8.36E-09
						1.39E-08

Notes:
ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-13

Summary of Potential Hazard Indices for Orlimulsion High Load Conditions
Resident at Saint John
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child			Adult		
	Soil	Water	Air	Soil	Water	Air
Mercury, elemental	9.71E-08	1.63E-07	7.39E-10	1.80E-09	NC	6.31E-10
Mercury, methyl	5.45E-06	6.36E-07	NC	1.01E-07	5.66E-08	NC
Mercury, divalent	3.60E-13	2.62E-10	4.81E-08	6.68E-15	2.21E-07	4.11E-08
1,4-Dichlorobenzene	4.04E-11	3.98E-10	3.53E-10	7.51E-13	9.13E-11	3.02E-10
Acenaphthene	6.27E-10	7.13E-09	3.69E-10	7.51E-13	1.39E-10	3.15E-10
Acenaphthylene	4.28E-12	5.55E-12	4.27E-12	1.16E-11	2.48E-09	3.66E-12
Anthracene	2.17E-07	6.66E-05	1.04E-07	7.95E-14	1.93E-12	3.65E-12
Arsenic	5.91E-12	2.12E-07	5.59E-07	4.04E-09	2.32E-05	8.86E-08
Benzene	1.97E-09	1.45E-09	2.62E-10	1.10E-13	7.38E-08	4.77E-07
Benzo(a)anthracene	3.04E-10	3.40E-10	7.55E-11	3.65E-11	5.06E-10	2.23E-10
Benzo(a)pyrene	1.48E-09	6.00E-10	2.15E-10	5.64E-12	1.18E-10	6.45E-11
Benzo(b)fluoranthene	2.80E-09	2.16E-09	2.09E-10	2.75E-11	2.09E-10	1.83E-10
Benzo(k)fluoranthene	5.60E-09	6.38E-08	5.74E-10	5.21E-11	7.52E-10	1.78E-10
Benzol(g,h,i)perylene	4.70E-08	6.31E-07	3.14E-07	1.04E-10	2.22E-08	4.90E-10
Beryllium	5.64E-09	6.78E-07	1.04E-09	8.74E-10	2.20E-07	2.68E-07
Cadmium	7.23E-09	8.87E-11	4.92E-11	1.05E-10	2.36E-07	8.91E-11
Chromium	2.68E-08	1.25E-05	7.38E-07	1.34E-10	3.09E-11	4.20E-11
Chromium VI	1.58E-09	1.78E-09	1.55E-10	4.98E-10	4.34E-06	6.31E-07
Chrysene	1.26E-09	7.76E-10	8.78E-11	2.93E-11	6.19E-10	1.32E-10
Dibenzo(a,h)anthracene	6.25E-10	5.91E-10	1.27E-10	2.34E-11	2.70E-10	7.50E-11
Fluoranthene	8.97E-11	8.35E-09	1.17E-10	1.16E-11	2.06E-10	1.09E-10
Fluorene	4.94E-14	6.49E-09	1.35E-08	1.67E-12	2.91E-09	9.98E-11
Formaldehyde	1.26E-09	4.01E-10	1.12E-10	9.17E-16	2.26E-09	1.16E-08
Indeno(1,2,3-cd)pyrene	8.30E-08	9.98E-07	6.15E-09	2.35E-11	1.40E-10	9.61E-11
Lead	1.79E-10	1.61E-08	2.52E-08	1.54E-09	3.47E-07	5.25E-09
Naphthalene	6.60E-06	9.13E-04	4.94E-03	3.32E-12	5.60E-09	2.16E-08
Nickel	4.26E-12	2.24E-09	2.75E-09	1.23E-07	3.18E-04	4.22E-03
Phenanthrene	1.40E-09	1.60E-08	1.44E-10	7.92E-14	7.80E-10	2.35E-09
Pyrene	2.46E-12	3.88E-08	9.67E-09	2.61E-11	5.57E-09	1.23E-10
Toluene	4.90E-05	7.95E-03	1.35E-05	4.58E-14	1.35E-08	8.26E-09
Vanadium	6.16E-05	8.87E-03	4.95E-03	9.10E-07	2.77E-03	1.16E-05
Total HI				1.14E-06	3.11E-03	4.23E-03

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-14

Summary of Potential Excess Lifetime Cancer Risk for Orlimulsion High Load Conditions

Resident at Saint John
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child					Adult				
	Soil	Water	Air	Produce	Risk	Soil	Water	Air	Produce	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	1.70E-16	1.24E-13	1.66E-13	2.27E-13	5.18E-13	1.58E-17	2.16E-13	7.09E-13	6.33E-13	1.56E-12
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	9.20E-12	2.83E-09	5.98E-11	5.65E-11	2.95E-09	8.55E-13	4.92E-09	2.56E-10	1.69E-10	5.35E-09
Benzene	4.41E-17	1.58E-12	2.36E-12	1.42E-13	4.08E-12	4.10E-18	2.75E-12	1.01E-11	4.02E-13	1.32E-11
Benzo(a)anthracene	7.12E-13	5.51E-13	1.11E-13	7.06E-14	1.44E-12	6.61E-14	9.59E-13	4.75E-13	2.11E-13	1.71E-12
Benzo(a)pyrene	1.11E-12	1.31E-12	3.21E-13	8.46E-13	3.59E-12	1.03E-13	2.28E-12	1.37E-12	2.54E-12	6.29E-12
Benzo(b)fluoranthene	5.39E-13	2.30E-13	9.13E-14	7.26E-14	9.32E-13	5.00E-14	4.00E-13	3.90E-13	2.17E-13	1.06E-12
Benzo(k)fluoranthene	8.39E-14	7.22E-14	8.88E-15	2.86E-14	1.94E-13	7.79E-15	1.26E-13	3.79E-14	8.58E-14	2.57E-13
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	ND	ND	1.29E-12	ND	1.29E-12	ND	ND	5.50E-12	ND	5.50E-12
Cadmium	ND	ND	2.52E-12	ND	2.52E-12	ND	ND	1.07E-11	ND	1.07E-11
Chromium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	4.87E-10	ND	4.87E-10	ND	ND	2.08E-09	ND	2.08E-09
Chrysene	5.48E-15	6.56E-15	6.57E-16	7.96E-16	1.35E-14	5.09E-16	1.14E-14	2.81E-15	2.38E-15	1.71E-14
Dibenzo(a,h)anthracene	4.36E-12	2.90E-12	3.73E-13	4.80E-12	1.24E-11	4.05E-13	5.05E-12	1.59E-12	1.44E-11	2.15E-11
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	1.04E-11	ND	1.04E-11	ND	ND	4.46E-11	ND	4.46E-11
Indeno(1,2,3-cd)pyrene	4.51E-13	1.53E-13	4.78E-14	1.10E-12	1.75E-12	4.18E-14	2.66E-13	2.04E-13	3.29E-12	3.81E-12
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	1.06E-08	ND	1.06E-08	ND	ND	4.51E-08	ND	4.51E-08
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total Risk	1.65E-11	4.67E-12	1.11E-08	6.38E-11	1.40E-08	1.53E-12	4.93E-09	4.75E-08	1.91E-10	5.26E-08

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-15
Summary of Potential Hazard Indices for Oromulsion High Load Conditions
Fisher Residing at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	HI	Soil	Water	Air	Produce	Fish	HI
Mercury, elemental	NC	NC	6.11E-12	NC	NC	6.11E-12	NC	NC	5.22E-12	NC	NC	5.22E-12
Mercury, methyl	2.45E-06	1.63E-07	NC	4.47E-07	1.57E-02	1.57E-02	4.55E-08	5.66E-08	NC	2.65E-07	2.10E-02	2.10E-02
Mercury, divalent	1.37E-04	6.36E-07	4.00E-10	4.46E-06	NC	1.42E-04	2.55E-06	2.21E-07	3.42E-10	2.62E-06	NC	5.40E-06
1,4-Dichlorobenzene	8.37E-12	2.62E-10	2.92E-12	1.12E-08	1.06E-09	1.25E-08	1.55E-13	9.13E-11	2.49E-12	6.24E-09	1.42E-09	7.75E-09
Acenaphthene	9.40E-10	3.98E-10	3.05E-12	1.81E-08	3.44E-09	2.28E-08	1.75E-11	1.39E-10	2.60E-12	1.01E-08	4.59E-09	1.49E-08
Acenaphthylene	2.57E-08	7.13E-09	3.66E-14	1.20E-08	1.21E-06	1.25E-06	4.78E-10	2.48E-09	3.12E-14	7.20E-09	1.61E-06	1.62E-06
Anthracene	9.95E-11	5.55E-12	3.53E-14	8.81E-11	2.05E-10	3.99E-10	1.85E-12	1.93E-12	3.01E-14	5.27E-11	2.74E-10	3.31E-10
Arsenic	1.28E-05	6.66E-05	1.07E-09	6.04E-05	1.90E-05	1.59E-04	2.37E-07	2.32E-05	9.17E-10	3.62E-05	2.53E-05	8.50E-05
Benzene	1.38E-10	2.12E-07	4.62E-09	4.42E-07	7.48E-08	7.34E-07	2.55E-12	7.38E-08	3.95E-09	2.51E-07	9.99E-08	4.29E-07
Benzo(a)anthracene	4.88E-08	1.45E-09	2.17E-12	5.86E-09	1.05E-07	1.62E-07	9.06E-10	5.06E-10	1.86E-12	3.51E-09	1.41E-07	1.46E-07
Benzo(a)pyrene	1.17E-08	3.40E-10	6.43E-13	8.15E-09	4.81E-08	6.83E-08	2.17E-10	1.18E-10	5.49E-13	4.89E-09	6.43E-08	6.95E-08
Benzo(b)fluoranthene	3.80E-08	6.00E-10	1.79E-12	5.96E-09	8.50E-08	1.30E-07	7.06E-10	2.09E-10	1.53E-12	3.57E-09	1.14E-07	1.18E-07
Benzo(k)fluoranthene	1.21E-07	2.16E-09	1.79E-12	2.76E-08	3.06E-07	4.57E-07	2.24E-09	7.52E-10	1.53E-12	1.65E-08	4.09E-07	4.28E-07
Benzo(g,h,i)perylene	2.30E-07	6.38E-08	4.91E-12	2.44E-08	1.08E-05	1.11E-05	4.27E-09	2.22E-08	4.19E-12	1.46E-08	1.44E-05	1.45E-05
Beryllium	2.76E-06	6.31E-07	3.25E-09	5.25E-07	5.57E-07	4.48E-06	5.13E-08	2.20E-07	2.77E-09	3.15E-07	7.44E-07	1.33E-06
Cadmium	3.31E-07	6.78E-07	1.08E-11	1.38E-06	2.41E-06	4.80E-06	6.16E-09	2.36E-07	9.22E-12	8.22E-07	3.22E-06	4.29E-06
Chromium	4.25E-07	8.87E-11	5.09E-13	3.04E-08	2.40E-10	4.56E-07	7.89E-09	3.09E-11	4.35E-13	1.82E-08	3.21E-10	2.65E-08
Chromium VI	1.58E-06	1.25E-05	7.64E-09	1.11E-05	5.33E-07	2.57E-05	2.93E-08	4.34E-06	5.53E-09	6.67E-06	7.11E-07	1.18E-05
Chrysene	4.19E-08	1.78E-09	1.29E-12	6.39E-09	1.53E-07	2.03E-07	7.79E-10	6.19E-10	1.10E-12	3.83E-09	2.04E-07	2.09E-07
Dibenzo(a,h)anthracene	7.35E-08	7.76E-10	9.06E-13	3.94E-08	1.41E-07	2.55E-07	1.36E-09	2.70E-10	7.74E-13	2.36E-08	1.89E-07	2.14E-07
Fluoranthene	1.46E-08	5.91E-10	1.05E-12	1.43E-09	1.32E-07	1.49E-07	2.71E-10	2.06E-10	8.98E-13	8.48E-10	1.76E-07	1.78E-07
Fluorene	2.16E-09	8.35E-09	9.68E-13	1.81E-09	1.43E-07	1.55E-07	4.01E-11	2.91E-09	8.27E-13	1.09E-09	1.91E-07	1.95E-07
Formaldehyde	1.15E-12	6.49E-09	1.12E-10	3.69E-08	9.88E-11	4.36E-08	2.13E-14	2.26E-09	9.56E-11	2.10E-08	1.32E-10	2.35E-08
Indeno(1,2,3-cd)pyrene	7.39E-08	4.01E-10	1.16E-12	5.05E-08	7.48E-08	2.00E-07	1.37E-09	1.40E-10	9.92E-13	3.03E-08	9.99E-08	1.32E-07
Lead	4.88E-06	9.98E-07	6.36E-11	1.01E-06	1.14E-07	7.00E-06	9.06E-08	3.47E-07	5.44E-11	6.05E-07	1.52E-07	1.19E-06
Naphthalene	4.16E-09	1.61E-08	2.09E-10	6.32E-07	4.92E-08	7.02E-07	7.73E-11	5.60E-09	1.78E-10	3.56E-07	6.57E-08	4.28E-07
Nickel	3.88E-04	9.13E-04	5.11E-05	8.66E-04	1.01E-03	3.23E-03	7.20E-06	3.18E-04	4.37E-05	5.19E-04	1.35E-03	2.24E-03
Phenanthrene	9.92E-11	2.24E-09	2.28E-11	3.66E-09	1.05E-07	1.11E-07	1.84E-12	7.80E-10	1.95E-11	2.19E-09	1.41E-07	1.44E-07
Pyrene	5.76E-08	1.60E-08	1.23E-12	2.69E-08	2.71E-06	2.81E-06	1.07E-09	5.57E-09	1.05E-12	1.61E-08	3.62E-06	3.64E-06
Toluene	5.73E-11	3.88E-08	8.00E-11	1.32E-07	3.46E-08	2.06E-07	1.06E-12	1.35E-08	6.83E-11	7.42E-08	4.62E-08	1.34E-07
Vanadium	2.88E-03	7.95E-03	1.41E-07	7.97E-03	1.13E-03	1.99E-02	5.35E-05	2.77E-03	1.20E-07	4.78E-03	1.51E-03	9.11E-03
Total HI	3.43E-03	8.87E-03	5.13E-05	8.91E-03	1.79E-02	3.93E-02	6.37E-05	3.11E-03	4.38E-05	5.35E-03	2.39E-02	3.25E-02

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-16

Summary of Potential Excess Lifetime Cancer Risk for Oritulmision High Load Conditions
 Fisher Residing at the Maximum Hypothetical Residence
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	Risk	Soil	Water	Air	Produce	Fish	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	3.96E-15	1.24E-13	1.37E-15	5.28E-12	5.01E-13	5.91E-12	3.68E-16	2.16E-13	5.87E-15	1.47E-11	3.35E-12	1.83E-11
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	5.41E-10	2.83E-09	6.19E-13	2.57E-09	8.05E-10	6.74E-09	5.02E-11	4.92E-09	2.64E-12	7.70E-09	5.38E-09	1.81E-08
Benzene	1.03E-15	1.58E-12	1.95E-14	3.30E-12	5.58E-13	5.46E-12	9.52E-17	2.75E-12	8.34E-14	9.35E-12	3.73E-12	1.59E-11
Benzol(a)anthracene	1.76E-11	5.51E-13	9.24E-16	2.26E-12	4.00E-11	6.05E-11	1.64E-12	9.59E-13	3.95E-15	6.76E-12	2.67E-10	2.77E-10
Benzol(a)pyrene	4.29E-11	1.31E-12	2.73E-15	3.21E-11	1.85E-10	2.62E-10	3.98E-12	2.28E-12	1.17E-14	9.62E-11	1.24E-09	1.34E-09
Benzol(b)fluoranthene	1.38E-11	2.30E-13	7.60E-16	2.33E-12	3.25E-11	4.89E-11	1.28E-12	4.00E-13	3.25E-15	6.98E-12	2.17E-10	2.26E-10
Benzol(k)fluoranthene	3.62E-12	7.22E-14	7.60E-17	1.07E-12	1.02E-11	1.50E-11	3.36E-13	1.26E-13	3.25E-16	3.20E-12	6.83E-11	7.20E-11
Benzol(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	ND	ND	1.33E-14	ND	ND	1.33E-14	ND	ND	5.69E-14	ND	ND	5.69E-14
Cadmium	ND	ND	2.60E-14	ND	ND	2.60E-14	ND	ND	1.11E-13	ND	ND	1.11E-13
Chromium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	5.04E-12	ND	ND	5.04E-12	ND	ND	2.15E-11	ND	ND	2.15E-11
Chrysene	1.46E-13	6.56E-15	5.49E-18	2.46E-14	5.63E-13	7.40E-13	1.35E-14	1.14E-14	2.34E-17	7.36E-14	3.76E-12	3.86E-12
Dibenzol(a,h)anthracene	2.54E-10	2.90E-12	3.85E-15	1.55E-10	5.29E-10	9.41E-10	2.36E-11	5.05E-12	1.65E-14	4.65E-10	3.53E-09	4.03E-09
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	8.63E-14	ND	ND	8.63E-14	ND	ND	3.69E-13	ND	ND	3.69E-13
Indeno(1,2,3-cd)pyrene	2.64E-11	1.53E-13	4.94E-16	1.99E-11	2.85E-11	7.49E-11	2.45E-12	2.66E-13	2.11E-15	5.97E-11	1.90E-10	2.53E-10
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	1.09E-10	ND	ND	1.09E-10	ND	ND	4.67E-10	ND	ND	4.67E-10
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total Risk	9.00E-10	4.67E-12	1.15E-10	2.79E-09	1.63E-09	8.27E-09	8.36E-11	4.93E-09	4.92E-10	8.36E-09	1.09E-08	2.48E-08

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-17

Summary of Potential Hazard Indices for Orimulsion High Load Conditions
 Fisher Residing at Saint John
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	HI	Soil	Water	Air	Produce	Fish	HI
Mercury, elemental	NC	NC	7.39E-10	NC	NC	7.39E-10	NC	NC	6.31E-10	NC	NC	6.31E-10
Mercury, methyl	9.71E-08	1.63E-07	NC	1.28E-08	1.57E-02	1.57E-02	1.80E-09	5.66E-08	NC	7.57E-09	2.10E-02	2.10E-02
Mercury, divalent	5.45E-06	6.36E-07	4.81E-08	2.07E-07	NC	6.34E-06	1.01E-07	2.21E-07	4.11E-08	1.22E-07	NC	4.86E-07
1,4-Dichlorobenzene	3.60E-13	2.62E-10	3.53E-10	4.82E-10	1.06E-09	2.16E-09	6.68E-15	9.13E-11	3.02E-10	2.68E-10	1.42E-09	2.08E-09
Acenaphthene	4.04E-11	3.98E-10	3.69E-10	7.79E-10	3.44E-09	5.02E-09	7.51E-13	1.39E-10	3.15E-10	4.36E-10	4.59E-09	5.48E-09
Acenaphthylene	6.27E-10	7.13E-09	4.28E-12	3.84E-10	1.21E-06	1.22E-06	1.16E-11	2.48E-09	3.66E-12	2.30E-10	1.61E-06	1.62E-06
Anthracene	4.28E-12	5.55E-12	4.27E-12	3.79E-12	2.05E-10	2.23E-10	7.95E-14	1.93E-12	3.65E-12	2.26E-12	2.74E-10	2.82E-10
Arsenic	2.17E-07	6.66E-05	1.04E-07	1.32E-06	1.90E-05	8.72E-05	4.04E-09	2.32E-05	8.86E-08	7.94E-07	2.53E-05	4.94E-05
Benzene	5.91E-12	2.12E-07	5.59E-07	1.90E-08	7.48E-08	8.65E-07	1.10E-13	7.38E-08	4.77E-07	1.08E-08	9.99E-08	6.62E-07
Benzo(a)anthracene	1.97E-09	1.45E-09	2.62E-10	1.85E-10	1.05E-07	1.09E-07	3.65E-11	5.06E-10	2.23E-10	1.10E-10	1.41E-07	1.42E-07
Benzo(a)pyrene	3.04E-10	3.40E-10	7.55E-11	2.15E-10	4.81E-08	4.90E-08	5.64E-12	1.18E-10	6.45E-11	1.29E-10	6.43E-08	6.46E-08
Benzo(b)fluoranthene	1.48E-09	6.00E-10	2.15E-10	1.86E-10	8.50E-08	8.75E-08	2.75E-11	2.09E-10	1.83E-10	1.11E-10	1.14E-07	1.14E-07
Benzo(k)fluoranthene	2.80E-09	2.16E-09	2.09E-10	7.38E-10	3.06E-07	3.12E-07	5.21E-11	7.52E-10	1.78E-10	4.42E-10	4.09E-07	4.10E-07
Benzo(g,h,i)perylene	5.60E-09	6.38E-08	5.74E-10	2.21E-09	1.08E-05	1.09E-05	1.04E-10	2.22E-08	4.90E-10	1.32E-09	1.44E-05	1.45E-05
Beryllium	4.70E-08	6.31E-07	3.14E-07	1.15E-08	5.57E-07	1.56E-06	8.74E-10	2.20E-07	2.68E-07	6.90E-09	7.44E-07	1.24E-06
Cadmium	5.64E-09	6.78E-07	1.04E-09	2.64E-08	2.41E-06	3.12E-06	1.05E-10	2.36E-07	8.91E-10	1.58E-08	3.22E-06	3.48E-06
Chromium	7.23E-09	8.87E-11	4.92E-11	6.58E-10	2.40E-10	8.27E-09	1.34E-10	3.09E-11	4.20E-11	3.95E-10	3.21E-10	9.22E-10
Chromium VI	2.68E-08	1.25E-05	7.38E-07	2.45E-07	5.33E-07	1.40E-05	4.98E-10	4.34E-06	6.31E-07	1.47E-07	7.11E-07	5.83E-06
Chrysene	1.58E-09	1.78E-09	1.55E-10	2.08E-10	1.53E-07	1.56E-07	2.93E-11	6.19E-10	1.32E-10	1.24E-10	2.04E-07	2.05E-07
Dibenzo(a,h)anthracene	1.26E-09	7.76E-10	8.78E-11	1.22E-09	1.41E-07	1.45E-07	2.34E-11	2.70E-10	7.50E-11	7.31E-10	1.89E-07	1.90E-07
Fluoranthene	6.25E-10	5.91E-10	1.27E-10	5.91E-11	1.32E-07	1.33E-07	1.16E-11	2.06E-10	1.09E-10	3.51E-11	1.76E-07	1.77E-07
Fluorene	8.97E-11	8.35E-09	1.17E-10	6.44E-11	1.43E-07	1.51E-07	1.67E-12	2.91E-09	9.98E-11	3.86E-11	1.91E-07	1.94E-07
Formaldehyde	4.94E-14	6.49E-09	1.35E-08	1.59E-09	9.88E-11	2.17E-08	9.17E-16	2.26E-09	1.16E-08	9.03E-10	1.32E-10	1.49E-08
Indeno(1,2,3-cd)pyrene	1.26E-09	4.01E-10	1.12E-10	2.79E-09	7.48E-08	7.94E-08	2.35E-11	1.40E-10	9.61E-11	1.67E-09	9.99E-08	1.02E-07
Lead	8.30E-08	9.98E-07	6.15E-09	2.13E-08	1.14E-07	1.22E-06	1.54E-09	3.47E-07	5.25E-09	1.28E-08	1.52E-07	5.19E-07
Naphthalene	1.79E-10	1.61E-08	2.52E-08	2.72E-08	4.92E-08	1.18E-07	3.32E-12	5.60E-09	2.16E-08	1.53E-08	6.57E-08	1.08E-07
Nickel	6.60E-06	9.13E-04	4.94E-03	1.88E-05	1.01E-03	6.89E-03	1.23E-07	3.18E-04	4.22E-03	1.13E-05	1.35E-03	5.90E-03
Phenanthrene	4.26E-12	2.24E-09	2.75E-09	1.57E-10	1.05E-07	1.10E-07	7.92E-14	7.80E-10	2.35E-09	9.43E-11	1.41E-07	1.44E-07
Pyrene	1.40E-09	1.60E-08	1.44E-10	8.60E-10	2.71E-06	2.73E-06	2.61E-11	5.57E-09	1.23E-10	5.15E-10	3.62E-06	3.62E-06
Toluene	2.46E-12	3.88E-08	9.67E-09	5.68E-09	3.46E-08	8.87E-08	4.58E-14	1.35E-08	8.26E-09	3.19E-09	4.62E-08	7.12E-08
Vanadium	4.90E-05	7.95E-03	1.36E-05	1.75E-04	1.13E-03	9.31E-03	9.10E-07	2.77E-03	1.16E-05	1.05E-04	1.51E-03	4.39E-03
Total HI	6.16E-05	8.87E-03	4.95E-03	1.95E-04	1.79E-02	3.20E-02	1.14E-06	3.11E-03	4.23E-03	1.17E-04	2.39E-02	3.14E-02

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-18

Summary of Potential Excess Lifetime Cancer Risk for Orimulsion High Load Conditions
 Fisher Residing at Saint John
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	Risk	Soil	Water	Air	Produce	Fish	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	1.70E-16	1.24E-13	1.66E-13	2.27E-13	5.01E-13	1.02E-12	1.58E-17	2.16E-13	7.09E-13	6.33E-13	3.35E-12	4.91E-12
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	9.20E-12	2.83E-09	5.98E-11	5.65E-11	8.05E-10	3.76E-09	8.55E-13	4.92E-09	2.56E-10	1.69E-10	5.38E-09	1.07E-08
Benzene	4.41E-17	1.58E-12	2.36E-12	1.42E-13	5.58E-13	4.64E-12	4.10E-18	2.75E-12	1.01E-11	4.02E-13	3.73E-12	1.70E-11
Benzofluoranthracene	7.12E-13	5.51E-13	1.11E-13	7.06E-14	4.00E-11	4.14E-11	6.61E-14	9.59E-13	4.75E-13	2.11E-13	2.67E-10	2.69E-10
Benzofluoranthene	1.11E-12	1.31E-12	3.21E-13	8.46E-13	1.85E-10	1.89E-10	1.03E-13	2.28E-12	1.37E-12	2.54E-12	1.24E-09	1.25E-09
Benzofluoranthene	5.39E-13	2.30E-13	9.13E-14	7.26E-14	3.25E-11	3.34E-11	5.00E-14	4.00E-13	3.90E-13	2.17E-13	2.17E-10	2.18E-10
Benzofluoranthene	8.39E-14	7.22E-14	8.88E-15	2.86E-14	1.02E-11	1.04E-11	7.79E-15	1.26E-13	7.79E-14	8.58E-14	6.83E-11	6.85E-11
Benzofluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Benzofluoranthene	ND	ND	1.29E-12	ND	ND	1.29E-12	ND	ND	5.50E-12	ND	ND	5.50E-12
Beryllium	ND	ND	2.52E-12	ND	ND	2.52E-12	ND	ND	1.07E-11	ND	ND	1.07E-11
Cadmium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chromium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	4.87E-10	ND	ND	4.87E-10	ND	ND	2.08E-09	ND	ND	2.08E-09
Chrysene	5.48E-15	6.56E-15	6.57E-16	7.95E-16	5.63E-13	5.76E-13	5.09E-16	1.14E-14	2.81E-15	2.38E-15	3.76E-12	3.78E-12
Dibenzofluoranthracene	4.36E-12	2.90E-12	3.73E-13	4.80E-12	5.29E-10	5.41E-10	4.05E-13	5.05E-12	1.59E-12	1.44E-11	3.53E-09	3.55E-09
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	1.04E-11	ND	ND	1.04E-11	ND	ND	4.46E-11	ND	ND	4.46E-11
Indeno(1,2,3-cd)pyrene	4.51E-13	1.53E-13	4.78E-14	1.10E-12	2.85E-11	3.03E-11	4.18E-14	2.66E-13	2.04E-13	3.29E-12	1.90E-10	1.94E-10
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	1.06E-08	ND	ND	1.06E-08	ND	ND	4.51E-08	ND	ND	4.51E-08
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total Risk	1.65E-11	4.67E-12	1.11E-08	6.38E-11	1.63E-09	1.57E-08	1.53E-12	4.93E-09	4.75E-08	1.91E-10	1.09E-08	6.35E-08

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-19
Summary of Potential Hazard Indices for Orimulsion High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child									
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	HI
Mercury, elemental	NC	NC	3.58E-10	NC	NC	NC	NC	NC	NC	3.58E-10
Mercury, methyl	5.23E-08	1.63E-07	NC	2.77E-08	1.27E-09	2.17E-08	1.31E-10	1.35E-10	3.04E-12	2.43E-07
Mercury, divalent	2.93E-06	6.36E-07	2.33E-08	4.36E-07	3.06E-07	4.08E-06	4.92E-08	5.07E-08	1.10E-09	4.03E-06
1,4-Dichlorobenzene	1.93E-13	2.62E-10	1.71E-10	1.03E-09	1.19E-14	3.08E-13	5.82E-16	2.40E-13	1.49E-14	1.47E-09
Acenaphthene	2.17E-11	3.98E-10	1.79E-10	1.67E-09	5.13E-13	9.06E-12	6.32E-15	2.61E-12	1.86E-13	2.27E-09
Acenaphthylene	3.48E-10	7.13E-09	2.09E-12	8.13E-10	2.94E-10	5.08E-09	5.04E-13	2.08E-10	2.84E-11	8.30E-09
Anthracene	2.30E-12	5.55E-12	2.07E-12	8.13E-12	9.82E-14	1.53E-12	1.25E-15	5.15E-13	3.32E-14	1.80E-11
Arsenic	1.11E-07	6.66E-05	5.07E-08	2.58E-06	2.24E-07	3.73E-07	NC	NC	NC	6.93E-05
Benzene	3.18E-12	2.12E-07	2.71E-07	4.08E-08	6.35E-14	1.69E-12	2.45E-15	1.01E-12	8.25E-14	5.24E-07
Benz(a)anthracene	1.06E-09	1.45E-09	1.27E-10	3.94E-10	6.81E-10	1.00E-08	7.10E-12	2.93E-09	1.92E-10	3.03E-09
Benz(a)pyrene	1.68E-10	3.40E-10	3.68E-11	4.61E-10	1.48E-09	2.54E-08	3.16E-12	1.30E-09	1.51E-10	1.00E-09
Benz(b)fluoranthene	8.00E-10	6.00E-10	1.04E-10	3.94E-10	2.47E-09	3.88E-08	1.78E-11	7.37E-09	5.34E-10	1.90E-09
Benz(k)fluoranthene	1.56E-09	2.16E-09	1.02E-10	1.58E-09	7.03E-09	1.15E-07	3.46E-11	1.43E-08	1.15E-09	5.41E-09
Benz(g,h,i)perylene	3.11E-09	6.38E-08	2.80E-10	4.24E-09	2.30E-09	3.96E-08	4.50E-12	1.86E-09	2.41E-10	7.14E-08
Beryllium	2.39E-08	6.31E-07	1.53E-07	2.24E-08	1.47E-09	6.34E-11	NC	NC	NC	8.31E-07
Cadmium	2.87E-09	6.78E-07	5.10E-10	5.25E-08	1.90E-10	5.83E-10	7.37E-10	1.79E-11	3.38E-11	7.34E-07
Chromium	3.68E-09	8.87E-11	2.40E-11	1.28E-09	7.20E-10	8.21E-09	NC	NC	NC	5.07E-09
Chromium VI	1.36E-08	1.25E-05	3.61E-07	4.77E-07	1.13E-07	1.72E-06	NC	NC	NC	1.33E-05
Chrysene	8.53E-10	1.78E-09	7.51E-11	4.41E-10	9.38E-10	1.48E-08	6.58E-12	2.71E-09	1.98E-10	3.15E-09
Dibenzo(a,h)anthracene	6.40E-10	7.76E-10	4.29E-11	2.37E-09	3.22E-08	5.61E-07	3.15E-11	1.30E-08	2.63E-09	3.83E-09
Fluoranthene	3.36E-10	5.91E-10	6.16E-11	1.27E-10	4.02E-11	5.50E-10	5.84E-13	2.41E-10	1.45E-11	1.11E-09
Fluorene	4.83E-11	8.35E-09	5.67E-11	1.36E-10	6.42E-12	1.11E-10	1.07E-14	4.42E-12	5.79E-13	8.59E-09
Formaldehyde	2.65E-14	6.49E-09	6.57E-09	3.41E-09	9.25E-17	2.46E-15	3.55E-18	1.47E-15	1.20E-16	1.65E-08
Indeno(1,2,3-cd)pyrene	6.43E-10	4.01E-10	5.50E-11	5.41E-09	2.98E-07	5.22E-06	7.38E-11	3.04E-08	2.05E-08	6.51E-09
Lead	4.22E-08	9.98E-07	3.01E-09	4.17E-08	8.89E-10	3.62E-08	NC	NC	1.97E-10	1.08E-06
Naphthalene	9.61E-11	1.61E-08	1.22E-08	5.84E-08	1.91E-12	4.27E-11	4.59E-14	1.90E-11	1.52E-12	8.68E-08
Nickel	3.55E-06	9.13E-04	2.41E-03	3.66E-05	9.46E-06	8.67E-05	NC	NC	NC	3.37E-03
Phenanthrene	2.29E-12	2.24E-09	1.34E-09	3.38E-10	4.55E-13	1.13E-11	1.68E-14	6.93E-12	5.56E-13	3.92E-09
Pyrene	7.80E-10	1.60E-08	7.01E-11	1.82E-09	6.59E-10	1.14E-08	1.13E-12	4.67E-10	6.37E-11	1.87E-08
Toluene	1.32E-12	3.88E-08	4.69E-09	1.22E-08	4.29E-14	1.13E-12	1.64E-15	6.75E-13	5.50E-14	5.57E-08
Vanadium	2.49E-05	7.95E-03	6.64E-06	3.40E-04	3.67E-05	1.62E-03	NC	NC	NC	8.32E-03
Total HI	3.15E-05	8.87E-03	2.42E-03	3.80E-04	4.72E-05	1.72E-03	5.02E-08	1.26E-07	2.71E-08	1.18E-02

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-19
Summary of Potential Hazard Indices for Orimulsion High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Adult									
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	HI
Mercury, elemental	NC	NC	3.06E-10	NC	2.45E-09	9.56E-09	1.63E-10	1.66E-10	NC	3.06E-10
Mercury, methyl	9.71E-10	5.66E-08	NC	1.64E-08	2.45E-09	9.56E-09	1.63E-10	1.66E-10	NC	3.06E-10
Mercury, divalent	5.45E-08	2.21E-07	1.99E-08	2.57E-07	5.94E-07	1.79E-06	6.12E-08	6.22E-08	1.27E-09	5.53E-07
1,4-Dichlorobenzene	3.59E-15	9.13E-11	1.46E-10	5.77E-10	2.30E-14	1.36E-13	7.24E-16	2.95E-13	1.72E-14	8.14E-10
Acenaphthene	4.03E-13	1.39E-10	1.53E-10	9.36E-10	9.93E-13	3.98E-12	7.86E-15	3.20E-12	2.15E-13	1.23E-09
Acenaphthylene	6.46E-12	2.48E-09	1.78E-12	4.87E-10	5.70E-10	2.24E-09	6.27E-13	2.55E-10	3.28E-11	2.98E-09
Anthracene	4.27E-14	1.93E-12	1.77E-12	4.86E-12	1.90E-13	6.71E-13	1.55E-15	6.32E-13	3.83E-14	8.60E-12
Arsenic	2.05E-09	2.32E-05	4.33E-08	1.55E-06	4.34E-07	1.64E-07	NC	NC	NC	2.48E-05
Benzene	5.90E-14	7.38E-08	2.32E-07	2.32E-08	1.23E-13	7.43E-13	3.04E-15	1.24E-12	9.52E-14	3.29E-07
Benzofluoranthracene	1.97E-11	5.06E-10	1.08E-10	2.35E-10	1.32E-09	4.41E-09	8.83E-12	3.59E-09	2.22E-10	8.70E-10
Benzofluoranthene	3.11E-12	1.18E-10	3.14E-11	2.76E-10	2.87E-09	1.12E-08	3.93E-12	1.60E-09	1.75E-10	4.29E-10
Benzofluoranthene	1.49E-11	2.09E-10	8.91E-11	2.36E-10	4.79E-09	1.71E-08	2.21E-11	9.05E-09	6.16E-10	5.49E-10
Benzofluoranthene	2.90E-11	7.52E-10	8.70E-11	9.47E-10	1.36E-08	5.04E-08	4.30E-11	1.75E-08	1.33E-09	1.82E-09
Benzofluoranthene	5.78E-11	2.22E-08	2.39E-10	2.54E-09	4.46E-09	1.74E-08	5.60E-12	2.28E-09	2.78E-10	2.50E-08
Beryllium	4.44E-10	2.20E-07	1.31E-07	1.34E-08	2.85E-09	2.79E-11	NC	NC	NC	3.65E-07
Cadmium	5.33E-11	2.36E-07	4.35E-10	3.14E-08	3.68E-10	2.56E-10	9.17E-10	2.20E-11	3.91E-11	2.68E-07
Chromium	6.83E-11	3.09E-11	2.05E-11	7.70E-10	1.40E-09	3.61E-09	NC	NC	NC	8.89E-10
Chromium VI	2.53E-10	4.34E-06	3.08E-07	2.86E-07	2.19E-07	7.55E-07	NC	NC	NC	4.94E-06
Chrysene	1.58E-11	6.19E-10	6.42E-11	2.64E-10	1.82E-09	6.49E-09	8.18E-12	3.32E-09	2.28E-10	9.63E-10
Dibenzofluoranthracene	1.19E-11	2.70E-10	3.66E-11	1.42E-09	6.23E-08	2.47E-07	3.92E-11	1.59E-08	3.04E-09	1.74E-09
Fluoranthene	6.23E-12	2.06E-10	5.27E-11	7.53E-11	7.79E-11	2.42E-10	7.26E-13	2.95E-10	1.68E-11	3.40E-10
Fluorene	8.96E-13	2.91E-09	4.84E-11	8.10E-11	1.24E-11	4.88E-11	1.33E-14	5.42E-12	6.88E-13	3.04E-09
Formaldehyde	4.92E-16	2.26E-09	5.61E-09	1.94E-09	1.79E-16	1.08E-15	4.42E-18	1.80E-15	1.38E-16	9.81E-09
Indeno(1,2,3-cd)pyrene	1.19E-11	1.40E-10	4.69E-11	3.25E-09	5.73E-07	2.29E-06	9.15E-11	3.73E-08	2.37E-08	3.45E-09
Lead	7.84E-10	3.47E-07	2.57E-09	2.50E-08	1.72E-09	1.59E-08	NC	NC	2.28E-10	3.76E-07
Naphthalene	1.78E-12	5.60E-09	1.05E-08	3.29E-08	3.70E-12	1.88E-11	5.71E-14	2.33E-11	1.75E-12	4.89E-08
Nickel	6.23E-08	3.18E-04	2.06E-03	2.20E-05	1.83E-05	3.81E-05	NC	NC	NC	2.40E-03
Phenanthrene	4.25E-14	7.80E-10	1.14E-09	2.02E-10	8.82E-13	4.99E-12	2.09E-14	8.50E-12	6.41E-13	2.12E-09
Pyrene	1.45E-11	5.57E-09	5.99E-11	1.09E-09	1.28E-09	5.01E-09	1.40E-12	5.72E-10	7.35E-11	6.73E-09
Toluene	2.46E-14	1.35E-08	4.01E-09	6.85E-09	8.31E-14	4.97E-13	2.03E-15	8.28E-13	6.34E-14	2.44E-08
Vanadium	4.63E-07	2.77E-03	5.67E-06	2.04E-04	7.11E-05	7.11E-04	NC	NC	NC	2.98E-03
Total HI	5.84E-07	3.11E-03	2.07E-03	2.28E-04	9.14E-05	7.55E-04	6.25E-08	1.54E-07	3.13E-08	5.41E-03

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

Table 5-20
Summary of Potential Excess Lifetime Cancer Risk for Orimulsion High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child									
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	9.14E-17	1.24E-13	8.06E-14	4.88E-13	5.60E-18	1.46E-16	2.75E-19	1.13E-16	7.04E-18	6.93E-13
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	4.68E-12	2.83E-09	2.92E-11	1.10E-10	9.60E-12	1.60E-11	NC	NC	NC	2.97E-09
Benzene	2.37E-17	1.58E-12	1.15E-12	3.04E-13	4.74E-19	1.26E-17	1.82E-20	7.54E-18	6.15E-19	3.03E-12
Benzo(a)anthracene	3.83E-13	5.51E-13	5.40E-14	1.51E-13	2.58E-13	3.83E-12	2.57E-15	1.06E-12	7.02E-14	1.14E-12
Benzo(a)pyrene	6.16E-13	1.31E-12	1.56E-13	1.81E-12	5.81E-12	9.98E-11	1.16E-14	4.79E-12	5.76E-13	3.90E-12
Benzo(b)fluoranthene	2.91E-13	2.30E-13	4.43E-14	1.54E-13	9.52E-13	1.51E-11	6.47E-15	2.68E-12	1.97E-13	7.18E-13
Benzo(k)fluoranthene	4.68E-14	7.22E-14	4.33E-15	6.13E-14	2.64E-13	4.38E-12	1.04E-15	4.28E-13	3.75E-14	1.85E-13
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	ND	ND	6.29E-13	ND	ND	ND	ND	ND	ND	6.29E-13
Cadmium	ND	ND	1.23E-12	ND	ND	ND	ND	ND	ND	1.23E-12
Chromium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	2.38E-10	ND	ND	ND	ND	ND	ND	2.38E-10
Chrysene	2.96E-15	6.56E-15	3.19E-16	1.69E-15	3.56E-15	5.66E-14	2.28E-17	9.39E-15	7.04E-16	1.15E-14
Dibenzo(a,h)anthracene	2.22E-12	2.90E-12	1.82E-13	9.33E-12	1.26E-10	2.21E-09	1.09E-13	4.50E-11	1.00E-11	1.46E-11
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	5.07E-12	ND	ND	ND	ND	ND	ND	5.07E-12
Indeno(1,2,3-cd)pyrene	2.29E-13	1.53E-13	2.34E-14	2.13E-12	1.17E-10	2.06E-09	2.62E-14	1.08E-11	8.03E-12	2.54E-12
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	5.16E-09	ND	ND	ND	ND	ND	ND	5.16E-09
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total Risk	8.47E-12	4.67E-12	5.44E-09	1.24E-10	2.60E-10	4.40E-09	1.57E-13	6.48E-11	1.89E-11	8.40E-09

Notes:
ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-20

Summary of Potential Excess Lifetime Cancer Risk, for Onimulsiom High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Adult										Risk
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	Risk	
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	8.49E-18	2.16E-13	3.44E-13	1.36E-12	5.42E-17	3.20E-16	1.71E-18	6.95E-16	4.06E-17	1.92E-12	ND
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	4.35E-13	4.92E-09	1.25E-10	3.29E-10	9.30E-11	3.51E-11	NC	NC	NC	5.38E-09	ND
Benzene	2.20E-18	2.75E-12	4.89E-12	8.64E-13	4.59E-18	2.77E-17	1.13E-19	4.62E-17	3.55E-18	8.51E-12	ND
Benzo(a)anthracene	3.56E-14	9.59E-13	2.31E-13	4.51E-13	2.50E-12	8.41E-12	1.60E-14	6.50E-12	4.05E-13	1.68E-12	ND
Benzo(a)pyrene	5.72E-14	2.28E-12	6.68E-13	5.44E-12	5.63E-11	2.19E-10	7.21E-14	2.94E-11	3.32E-12	8.44E-12	ND
Benzo(b)fluoranthene	2.70E-14	4.00E-13	1.89E-13	4.61E-13	9.22E-12	3.32E-11	4.02E-14	1.64E-11	1.14E-12	1.08E-12	ND
Benzo(k)fluoranthene	4.34E-15	1.26E-13	1.85E-14	1.84E-13	2.55E-12	9.63E-12	6.44E-15	2.63E-12	2.16E-13	3.32E-13	ND
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	ND	ND	2.69E-12	ND	ND	ND	ND	ND	ND	2.69E-12	ND
Cadmium	ND	ND	5.25E-12	ND	ND	ND	ND	ND	ND	5.25E-12	ND
Chromium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chromium VI	ND	ND	1.02E-09	ND	ND	ND	ND	ND	ND	1.02E-09	ND
Chrysene	2.75E-16	1.14E-14	1.36E-15	5.05E-15	3.45E-14	1.24E-13	1.42E-16	5.76E-14	4.06E-15	1.81E-14	ND
Dibenz(a,h)anthracene	2.06E-13	5.05E-12	7.79E-13	2.80E-11	1.22E-09	4.85E-09	6.79E-13	2.78E-10	5.78E-11	3.40E-11	ND
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	2.16E-11	ND	ND	ND	ND	ND	ND	2.16E-11	ND
Indeno(1,2,3-cd)pyrene	2.13E-14	2.66E-13	9.98E-14	6.40E-12	1.13E-09	4.52E-09	1.63E-13	6.64E-11	4.63E-11	6.79E-12	ND
Lead	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	2.20E-08	ND	ND	ND	ND	ND	ND	2.20E-08	ND
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Total Risk	7.86E-13	4.93E-09	2.32E-08	3.73E-10	2.51E-09	9.67E-09	9.77E-13	3.97E-10	1.09E-10	2.85E-08	ND

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

Table 5-21

Summary of Potential Hazard Indices for Natural Gas High Load Conditions
Resident at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child				Adult			
	Soil	Water	Air	HI	Soil	Water	Air	HI
Mercury, elemental	NC	NC	1.15E-13	1.15E-13	NC	NC	9.85E-14	9.85E-14
Mercury, methyl	3.64E-08	2.59E-09	NC	4.56E-08	6.75E-10	9.01E-10	NC	3.93E-09
Mercury, divalent	2.04E-06	1.02E-08	7.55E-12	2.12E-06	3.79E-08	3.55E-09	6.45E-12	3.89E-08
1,4-Dichlorobenzene	5.79E-11	2.29E-09	2.57E-11	7.75E-08	1.08E-12	7.97E-10	2.19E-11	4.32E-08
Acenaphthene	3.55E-11	1.83E-11	1.46E-13	6.83E-10	6.60E-13	6.39E-12	1.25E-13	3.82E-10
Acenaphthylene	8.07E-08	2.60E-08	1.46E-13	3.77E-08	1.50E-09	9.06E-09	1.25E-13	2.26E-08
Anthracene	8.66E-11	5.72E-12	3.90E-14	7.66E-11	1.61E-12	1.99E-12	3.33E-14	4.58E-11
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	1.41E-10	2.75E-07	6.01E-09	7.34E-07	2.62E-12	9.57E-08	5.13E-09	2.57E-07
Benzo(a)anthracene	9.69E-09	3.26E-10	5.49E-13	1.16E-09	1.80E-10	1.14E-10	4.69E-13	6.95E-10
Benzo(a)pyrene	5.22E-09	1.81E-10	3.67E-13	3.64E-09	9.70E-11	6.30E-11	3.13E-13	2.18E-09
Benzo(b)fluoranthene	9.17E-09	1.67E-10	5.49E-13	1.43E-09	1.70E-10	5.81E-11	4.69E-13	8.59E-10
Benzo(k)fluoranthene	2.91E-08	6.12E-10	5.49E-13	6.64E-09	5.40E-10	2.13E-10	4.69E-13	3.98E-09
Benzo(g,h,i)perylene	5.39E-08	1.74E-08	1.47E-12	5.73E-09	1.00E-09	6.05E-09	1.25E-12	3.42E-09
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	1.40E-08	6.67E-10	5.49E-13	2.13E-09	2.60E-10	2.32E-10	4.69E-13	1.28E-09
Dibenzo(a,h)anthracene	2.31E-08	2.84E-10	3.67E-13	1.24E-08	4.30E-10	9.88E-11	3.13E-13	7.44E-09
Fluoranthene	3.99E-09	1.80E-10	3.65E-13	3.90E-10	7.42E-11	6.27E-11	3.12E-13	2.32E-10
Fluorene	5.99E-10	2.57E-09	3.41E-13	5.02E-10	1.11E-11	8.95E-10	2.91E-13	3.01E-10
Formaldehyde	1.48E-11	1.05E-07	1.83E-09	4.75E-07	2.74E-13	3.65E-08	1.56E-09	2.70E-07
Indeno(1,2,3-cd)pyrene	2.72E-08	1.72E-10	5.49E-13	1.86E-08	5.05E-10	5.98E-11	4.69E-13	1.11E-08
Lead	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	2.92E-09	1.41E-08	1.86E-10	4.44E-07	5.42E-11	4.90E-09	1.59E-10	2.50E-07
Nickel	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	7.12E-11	2.04E-09	2.07E-11	2.63E-09	1.32E-12	7.09E-10	1.77E-11	1.57E-09
Pyrene	2.99E-08	9.63E-09	8.13E-13	1.40E-08	5.55E-10	3.35E-09	6.94E-13	8.36E-09
Toluene	7.48E-12	6.40E-09	1.32E-11	1.72E-08	1.39E-13	2.23E-09	1.13E-11	9.69E-09
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA
Total HI	2.37E-06	1.59E-07	8.10E-09	1.65E-06	4.40E-08	1.66E-07	6.92E-09	9.37E-07

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-22

Summary of Potential Excess Lifetime Cancer Risk for Natural Gas High Load Conditions
Resident at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child				Adult			
	Soil	Water	Air	Produce	Risk	Soil	Water	Produce
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	2.74E-14	1.08E-12	1.21E-14	3.66E-11	3.77E-11	2.54E-15	1.89E-12	1.02E-10
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	1.05E-15	2.05E-12	2.54E-14	3.38E-12	5.45E-12	9.76E-17	3.57E-12	9.58E-12
Benz(a)anthracene	3.51E-12	1.24E-13	2.33E-16	4.48E-13	4.08E-12	3.26E-13	2.16E-13	1.34E-12
Benz(a)pyrene	1.92E-11	6.99E-13	1.56E-15	1.43E-11	3.42E-11	1.78E-12	1.22E-12	4.30E-11
Benz(b)fluoranthene	3.33E-12	6.41E-14	2.33E-16	5.61E-13	3.96E-12	3.10E-13	1.12E-13	1.68E-12
Benz(k)fluoranthene	8.70E-13	2.05E-14	2.33E-17	2.57E-13	1.15E-12	8.08E-14	3.57E-14	7.70E-13
Benzol(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	4.87E-14	2.47E-15	2.33E-18	8.20E-15	5.94E-14	4.52E-15	4.30E-15	2.45E-14
Dibenzo(a,h)anthracene	8.01E-11	1.06E-12	1.56E-15	4.88E-11	1.30E-10	7.44E-12	1.85E-12	1.46E-10
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	1.41E-12	ND	1.41E-12	ND	ND	ND
Indeno(1,2,3-cd)pyrene	9.69E-12	6.55E-14	2.33E-16	7.31E-12	1.71E-11	9.00E-13	1.14E-13	2.19E-11
Lead	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA
Total Risk	1.17E-10	1.91E-12	1.45E-12	1.12E-10	2.35E-10	1.08E-11	9.00E-12	3.27E-10

Notes

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.
NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-23
Summary of Potential Hazard Indices for Natural Gas High Load Conditions
Resident at Saint John
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child				Adult			
	Soil	Water	Air	HI	Soil	Water	Air	Produce
Mercury, elemental	NC	NC	5.19E-12	5.19E-12	NC	NC	4.43E-12	NC
Mercury, methyl	1.15E-09	2.59E-09	NC	3.91E-09	2.13E-11	9.01E-10	NC	1.02E-10
Mercury, divalent	6.45E-08	1.02E-08	3.37E-10	7.73E-08	1.20E-09	3.55E-09	2.88E-10	1.35E-09
1,4-Dichlorobenzene	1.93E-12	2.29E-09	1.15E-09	6.03E-09	3.58E-14	7.97E-10	9.86E-10	1.44E-09
Acenaphthene	1.18E-12	1.83E-11	6.58E-12	4.89E-11	2.20E-14	6.39E-12	5.62E-12	1.28E-11
Acenaphthylene	1.92E-09	2.60E-08	6.35E-12	2.90E-08	3.56E-11	9.06E-09	5.42E-12	6.17E-10
Anthracene	2.88E-12	5.72E-12	1.75E-12	1.29E-11	5.36E-14	1.99E-12	1.50E-12	1.53E-12
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	4.69E-12	2.75E-07	2.70E-07	5.60E-07	8.71E-14	9.57E-08	2.31E-07	8.56E-09
Benzo(a)anthracene	3.10E-10	3.26E-10	2.46E-11	6.92E-10	5.75E-12	1.14E-10	2.10E-11	1.89E-11
Benzo(a)pyrene	1.29E-10	1.81E-10	1.60E-11	4.15E-10	2.39E-12	6.30E-11	1.36E-11	5.39E-11
Benzo(b)fluoranthene	2.87E-10	1.67E-10	2.45E-11	5.17E-10	5.33E-12	5.81E-11	2.09E-11	2.32E-11
Benzo(k)fluoranthene	6.74E-10	6.12E-10	2.38E-11	1.47E-09	1.25E-11	2.13E-10	2.03E-11	9.88E-11
Benzo(g,h,i)perylene	1.28E-09	1.74E-08	6.36E-11	1.90E-08	2.38E-11	6.05E-09	5.43E-11	1.92E-10
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	4.29E-10	6.67E-10	2.44E-11	1.18E-09	7.96E-12	2.32E-10	2.09E-11	3.53E-11
Dibenzo(a,h)anthracene	3.72E-10	2.84E-10	1.32E-11	9.52E-10	6.91E-12	9.88E-11	1.13E-11	1.70E-10
Fluoranthene	1.33E-10	1.80E-10	1.64E-11	3.42E-10	2.46E-12	6.27E-11	1.40E-11	7.53E-12
Fluorene	1.95E-11	2.57E-09	1.53E-11	2.62E-09	3.62E-13	8.95E-10	1.31E-11	8.77E-12
Formaldehyde	4.92E-13	1.05E-07	8.22E-08	2.03E-07	9.13E-15	3.65E-08	7.03E-08	8.99E-09
Indeno(1,2,3-cd)pyrene	4.36E-10	1.72E-10	1.98E-11	1.27E-09	8.10E-12	5.98E-11	1.69E-11	3.83E-10
Lead	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	9.73E-11	1.41E-08	8.36E-09	3.73E-08	1.81E-12	4.90E-09	7.14E-09	8.32E-09
Nickel	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	2.37E-12	2.04E-09	9.34E-10	3.06E-09	4.40E-14	7.09E-10	7.98E-10	5.24E-11
Pyrene	7.11E-10	9.63E-09	3.53E-11	1.08E-08	1.32E-11	3.35E-09	3.01E-11	2.28E-10
Toluene	2.49E-13	6.40E-09	5.96E-10	7.57E-09	4.63E-15	2.23E-09	5.09E-10	3.23E-10
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA
Total HI	7.24E-08	1.59E-07	3.64E-07	5.45E-08	1.35E-09	1.66E-07	3.11E-07	3.10E-08

Notes:
NC - Not calculated. This compound is not assumed to be present in this medium.
NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-24

Summary of Potential Excess Lifetime Cancer Risk for Natural Gas High Load Conditions
Resident at Saint John
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child				Adult					
	Soil	Water	Air	Produce	Risk	Soil	Water	Air	Produce	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	9.13E-16	1.08E-12	5.43E-13	1.22E-12	2.85E-12	8.48E-17	1.89E-12	2.32E-12	3.39E-12	7.60E-12
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	3.50E-17	2.05E-12	1.14E-12	1.12E-13	3.30E-12	3.25E-18	3.57E-12	4.88E-12	3.19E-13	8.77E-12
Benz(a)anthracene	1.12E-13	1.24E-13	1.04E-14	1.21E-14	2.59E-13	1.04E-14	2.16E-13	4.46E-14	3.63E-14	3.07E-13
Benz(a)pyrene	4.72E-13	6.99E-13	6.78E-14	3.54E-13	1.59E-12	4.38E-14	1.22E-12	2.90E-13	1.06E-12	2.61E-12
Benz(b)fluoranthene	1.04E-13	6.41E-14	1.04E-14	1.51E-14	1.94E-13	9.68E-15	1.12E-13	4.45E-14	4.53E-14	2.11E-13
Benz(k)fluoranthene	2.02E-14	2.05E-14	1.01E-15	6.39E-15	4.81E-14	1.87E-15	3.57E-14	4.31E-15	1.91E-14	6.11E-14
Benz(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	1.49E-15	2.47E-15	1.04E-16	2.26E-16	4.29E-15	1.38E-16	4.30E-15	4.44E-16	6.77E-16	5.56E-15
Dibenz(a,h)anthracene	1.29E-12	1.06E-12	5.62E-14	1.11E-12	3.52E-12	1.20E-13	1.85E-12	2.40E-13	3.34E-12	5.55E-12
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	6.34E-11	ND	6.34E-11	ND	ND	2.71E-10	ND	2.71E-10
Indeno(1,2,3-cd)pyrene	1.55E-13	6.55E-14	8.41E-15	2.52E-13	4.81E-13	1.44E-14	1.14E-13	3.59E-14	7.55E-13	9.20E-13
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total Risk	2.15E-12	1.91E-12	6.53E-11	3.08E-12	7.57E-11	2.00E-13	9.00E-12	2.79E-10	8.97E-12	2.97E-10

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-25
Summary of Potential Hazard Indices for Natural Gas High Load Conditions
Fisher Residing at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	HI	Soil	Water	Air	Produce	Fish	HI
Mercury, elemental	NC	NC	1.15E-13	NC	NC	1.15E-13	NC	NC	9.85E-14	NC	NC	9.85E-14
Mercury, methyl	3.64E-08	2.59E-09	NC	6.62E-09	2.50E-04	2.51E-04	6.75E-10	9.01E-10	NC	3.93E-09	3.35E-04	3.35E-04
Mercury, divalent	2.04E-06	1.02E-08	7.55E-12	6.61E-08	NC	2.12E-06	3.79E-08	3.55E-09	6.45E-12	3.89E-08	NC	8.04E-08
1,4-Dichlorobenzene	5.79E-11	2.29E-09	2.57E-11	7.75E-08	9.25E-09	8.91E-08	1.08E-12	7.97E-10	2.19E-11	4.32E-08	1.24E-08	5.64E-08
Acenaphthene	3.55E-11	1.83E-11	1.46E-13	6.83E-10	1.58E-10	8.95E-10	6.60E-13	6.39E-12	1.25E-13	3.82E-10	2.12E-10	6.01E-10
Acenaphthylene	8.07E-08	2.60E-08	1.46E-13	3.77E-08	4.40E-06	4.55E-06	1.50E-09	9.06E-09	1.25E-13	2.26E-08	5.89E-06	5.92E-06
Anthracene	8.66E-11	5.72E-12	3.90E-14	7.66E-11	2.12E-10	3.81E-10	1.61E-12	1.99E-12	3.33E-14	4.58E-11	2.83E-10	3.32E-10
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	1.41E-10	2.75E-07	6.01E-09	4.53E-07	9.70E-08	8.31E-07	2.62E-12	9.57E-08	5.13E-09	2.57E-07	1.30E-07	4.87E-07
Benzo(a)anthracene	9.69E-09	3.26E-10	5.49E-13	1.16E-09	2.37E-08	3.48E-08	1.80E-10	1.14E-10	4.69E-13	6.95E-10	3.16E-08	3.26E-08
Benzo(a)pyrene	5.22E-09	1.81E-10	3.67E-13	3.64E-09	2.56E-08	3.47E-08	9.70E-11	6.30E-11	3.13E-13	2.18E-09	3.42E-08	3.66E-08
Benzo(b)fluoranthene	9.17E-09	1.67E-10	5.49E-13	1.43E-09	2.36E-08	3.44E-08	1.70E-10	5.81E-11	4.69E-13	8.59E-10	3.16E-08	3.27E-08
Benzo(k)fluoranthene	2.91E-08	6.12E-10	5.49E-13	6.64E-09	8.66E-08	1.23E-07	5.40E-10	2.13E-10	4.69E-13	3.98E-09	1.16E-07	1.20E-07
Benzo(g,h,i)perylene	5.39E-08	1.74E-08	1.47E-12	5.73E-09	2.94E-06	3.02E-06	1.00E-09	6.05E-09	1.25E-12	3.42E-09	3.93E-06	3.94E-06
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	1.40E-08	6.67E-10	5.49E-13	2.13E-09	5.72E-08	7.41E-08	2.60E-10	2.32E-10	4.69E-13	1.28E-09	7.65E-08	7.82E-08
Dibenzo(a,h)anthracene	2.31E-08	2.84E-10	3.67E-13	1.24E-08	5.17E-08	8.75E-08	4.30E-10	9.88E-11	3.13E-13	7.44E-09	6.90E-08	7.70E-08
Fluoranthene	3.99E-09	1.80E-10	3.65E-13	3.90E-10	4.02E-08	4.48E-08	7.42E-11	6.27E-11	3.12E-13	2.32E-10	5.37E-08	5.41E-08
Fluorene	5.99E-10	2.57E-09	3.41E-13	5.02E-10	4.39E-08	4.76E-08	1.11E-11	8.95E-10	2.91E-13	3.01E-10	5.87E-08	5.99E-08
Formaldehyde	1.48E-11	1.05E-07	1.83E-09	4.75E-07	1.60E-09	5.83E-07	2.74E-13	3.65E-08	1.56E-09	2.70E-07	2.14E-09	3.10E-07
Indeno(1,2,3-cd)pyrene	2.72E-08	1.72E-10	5.49E-13	1.86E-08	3.20E-08	7.79E-08	5.05E-10	5.98E-11	4.69E-13	1.11E-08	4.28E-08	5.45E-08
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	2.92E-09	1.41E-08	1.86E-10	4.44E-07	4.31E-08	5.04E-07	5.42E-11	4.90E-09	1.59E-10	2.50E-07	5.76E-08	3.12E-07
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	7.12E-11	2.04E-09	2.07E-11	2.63E-09	9.56E-08	1.00E-07	1.32E-12	7.09E-10	1.77E-11	1.57E-09	1.28E-07	1.30E-07
Pyrene	2.99E-08	9.63E-09	8.13E-13	1.40E-08	1.63E-06	1.68E-06	5.55E-10	3.35E-09	6.94E-13	8.36E-09	2.18E-06	2.19E-06
Toluene	7.48E-12	6.40E-09	1.32E-11	1.72E-08	5.71E-09	2.94E-08	1.39E-13	2.23E-09	1.13E-11	9.69E-09	7.63E-09	1.96E-08
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total HI	2.37E-06	1.59E-07	8.10E-09	1.65E-06	2.60E-04	2.65E-04	4.40E-08	1.66E-07	6.92E-09	9.37E-07	3.47E-04	3.49E-04

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-26
Summary of Potential Excess Lifetime Cancer Risk for Natural Gas High Load Conditions
Fisher Residing at the Maximum Hypothetical Residence
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	Risk	Soil	Water	Air	Produce	Fish	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	2.74E-14	1.08E-12	1.21E-14	3.66E-11	4.38E-12	4.21E-11	2.54E-15	1.89E-12	5.15E-14	1.02E-10	2.92E-11	1.33E-10
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	1.05E-15	2.05E-12	2.54E-14	3.38E-12	7.23E-13	6.18E-12	9.76E-17	3.57E-12	1.08E-13	9.58E-12	4.83E-12	1.81E-11
Benzo(a)anthracene	3.51E-12	1.24E-13	2.33E-16	4.48E-13	9.00E-12	1.31E-11	3.26E-13	2.16E-13	9.96E-16	1.34E-12	6.01E-11	6.20E-11
Benzo(a)pyrene	1.92E-11	6.99E-13	1.56E-15	1.43E-11	9.89E-11	1.33E-10	1.78E-12	1.22E-12	6.66E-15	4.30E-11	6.61E-10	7.07E-10
Benzo(b)fluoranthene	3.33E-12	6.41E-14	2.33E-16	5.61E-13	9.07E-12	1.30E-11	3.10E-13	1.12E-13	9.96E-16	1.68E-12	6.08E-11	6.27E-11
Benzo(k)fluoranthene	8.70E-13	2.05E-14	2.33E-17	2.57E-13	2.91E-12	4.05E-12	8.08E-14	3.57E-14	9.96E-17	7.70E-13	1.94E-11	2.03E-11
Benzo(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	4.87E-14	2.47E-15	2.33E-18	8.20E-15	2.12E-13	2.71E-13	4.52E-15	4.30E-15	9.96E-18	2.45E-14	1.42E-12	1.45E-12
Dibenzo(a,h)anthracene	8.01E-11	1.06E-12	1.56E-15	4.88E-11	1.94E-10	3.24E-10	7.44E-12	1.85E-12	6.66E-15	1.46E-10	1.29E-09	1.45E-09
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	1.41E-12	ND	ND	1.41E-12	ND	ND	6.02E-12	ND	ND	6.02E-12
Indeno(1,2,3-cd)pyrene	9.69E-12	6.55E-14	2.33E-16	7.31E-12	1.22E-11	2.93E-11	9.00E-13	1.14E-13	9.96E-16	2.19E-11	8.16E-11	1.05E-10
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total Risk	1.17E-10	1.91E-12	1.45E-12	1.12E-10	3.31E-10	5.66E-10	1.08E-11	9.00E-12	6.20E-12	3.27E-10	2.21E-09	2.56E-09

Notes:
ND - Not determined because this compound is not considered carcinogenic by this route of exposure.
NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-27

Summary of Potential Hazard Indices for Natural Gas High Load Conditions
 Fisher Residing at Saint John
 Human Health Risk Assessment
 Colleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	HI	Soil	Water	Air	Produce	Fish	HI
Mercury, elemental	NC	NC	5.19E-12	NC	NC	5.19E-12	NC	NC	4.43E-12	NC	NC	4.43E-12
Mercury, methyl	1.15E-09	2.59E-09	NC	1.72E-10	2.50E-04	2.50E-04	2.13E-11	9.01E-10	NC	1.02E-10	3.35E-04	3.35E-04
Mercury, divalent	6.45E-08	1.02E-08	3.37E-10	2.29E-09	NC	7.73E-08	1.20E-09	3.55E-09	2.88E-10	1.35E-09	NC	6.39E-09
1,4-Dichlorobenzene	1.93E-12	2.29E-09	1.15E-09	2.58E-09	9.25E-09	1.53E-08	3.58E-14	7.97E-10	9.86E-10	1.44E-09	1.24E-08	1.56E-08
Acenaphthene	1.18E-12	1.83E-11	6.58E-12	2.28E-11	1.58E-10	2.07E-10	2.20E-14	6.39E-12	5.62E-12	1.28E-11	2.12E-10	2.36E-10
Acenaphthylene	1.92E-09	2.60E-08	6.35E-12	1.03E-09	4.40E-06	4.43E-06	3.56E-11	9.06E-09	5.42E-12	6.17E-10	5.88E-06	5.89E-06
Anthracene	2.88E-12	5.72E-12	1.75E-12	2.55E-12	2.12E-10	2.25E-10	5.36E-14	1.99E-12	1.50E-12	1.53E-12	2.83E-10	2.88E-10
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	4.69E-12	2.75E-07	2.70E-07	1.51E-08	9.70E-08	6.57E-07	8.71E-14	9.57E-08	2.31E-07	8.56E-09	1.30E-07	4.65E-07
Benzo(a)anthracene	3.10E-10	3.26E-10	2.46E-11	3.17E-11	2.37E-08	2.44E-08	5.75E-12	1.14E-10	2.10E-11	1.89E-11	3.16E-08	3.18E-08
Benzo(a)pyrene	1.29E-10	1.81E-10	1.60E-11	8.98E-11	2.56E-08	2.60E-08	2.39E-12	6.30E-11	1.36E-11	5.39E-11	3.42E-08	3.44E-08
Benzo(b)fluoranthene	2.87E-10	1.67E-10	2.45E-11	3.87E-11	2.36E-08	2.42E-08	5.33E-12	5.81E-11	2.09E-11	2.32E-11	3.16E-08	3.17E-08
Benzo(k)fluoranthene	6.74E-10	6.12E-10	2.38E-11	1.65E-10	8.66E-08	8.81E-08	1.25E-11	2.13E-10	2.03E-11	9.88E-11	1.16E-07	1.16E-07
Benzo(g,h,i)perylene	1.28E-09	1.74E-08	6.36E-11	3.21E-10	2.94E-06	2.96E-06	2.38E-11	6.05E-09	5.43E-11	1.92E-10	3.93E-06	3.94E-06
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	4.29E-10	6.67E-10	2.44E-11	5.90E-11	5.72E-08	5.84E-08	7.96E-12	2.32E-10	2.09E-11	3.53E-11	7.65E-08	7.68E-08
Dibenzo(a,h)anthracene	3.72E-10	2.84E-10	1.32E-11	2.83E-10	5.17E-08	5.26E-08	6.91E-12	9.88E-11	1.13E-11	1.70E-10	6.90E-08	6.93E-08
Fluoranthene	1.33E-10	1.80E-10	1.64E-11	1.27E-11	4.02E-08	4.06E-08	2.46E-12	6.27E-11	1.40E-11	7.53E-12	5.37E-08	5.38E-08
Fluorene	1.95E-11	2.57E-09	1.53E-11	1.46E-11	4.39E-08	4.65E-08	3.62E-13	8.95E-10	1.31E-11	8.77E-12	5.87E-08	5.96E-08
Formaldehyde	4.92E-13	1.05E-07	8.22E-08	1.58E-08	1.60E-09	2.05E-07	9.13E-15	3.65E-08	7.03E-08	8.99E-09	2.14E-09	1.18E-07
Indeno(1,2,3-cd)pyrene	4.36E-10	1.72E-10	1.98E-11	6.39E-10	3.20E-08	3.33E-08	8.10E-12	5.98E-11	1.69E-11	3.83E-10	4.28E-08	4.33E-08
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	9.73E-11	1.41E-08	8.36E-09	1.48E-08	4.31E-08	8.04E-08	1.81E-12	4.90E-09	7.14E-09	8.32E-09	5.76E-08	7.79E-08
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	2.37E-12	2.04E-09	9.34E-10	8.75E-11	9.56E-08	9.86E-08	4.40E-14	7.09E-10	7.98E-10	5.24E-11	1.28E-07	1.29E-07
Pyrene	7.11E-10	9.63E-09	3.53E-11	3.81E-10	1.63E-06	1.64E-06	1.32E-11	3.35E-09	3.01E-11	2.28E-10	2.18E-06	2.18E-06
Toluene	2.49E-13	6.40E-09	5.96E-10	5.74E-10	5.71E-09	1.33E-08	4.63E-15	2.23E-09	5.09E-10	3.23E-10	7.63E-09	1.07E-08
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total HI	7.24E-08	1.59E-07	3.64E-07	5.45E-08	2.60E-04	2.61E-04	1.35E-09	1.66E-07	3.11E-07	3.10E-08	3.47E-04	3.48E-04

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-28

Summary of Potential Excess Lifetime Cancer Risk for Natural Gas High Load Conditions
 Fisher Residing at Saint John
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Compound	Child						Adult					
	Soil	Water	Air	Produce	Fish	Risk	Soil	Water	Air	Produce	Fish	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	9.13E-16	1.08E-12	5.43E-13	1.22E-12	4.38E-12	7.22E-12	8.48E-17	1.89E-12	2.32E-12	3.39E-12	2.92E-11	3.68E-11
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	3.50E-17	2.05E-12	1.14E-12	1.12E-13	7.23E-13	4.03E-12	3.25E-18	3.57E-12	4.88E-12	3.19E-13	4.83E-12	1.36E-11
Benzofluoranthracene	1.12E-13	1.24E-13	1.04E-14	1.21E-14	9.00E-12	9.26E-12	1.04E-14	2.16E-13	4.46E-14	3.63E-14	6.01E-11	6.04E-11
Benzofluoranthene	4.72E-13	6.99E-13	6.78E-14	3.54E-13	9.89E-11	1.01E-10	4.38E-14	1.22E-12	2.90E-13	1.06E-12	6.61E-10	6.64E-10
Benzofluoranthene	1.04E-13	6.41E-14	1.04E-14	1.51E-14	9.07E-12	9.27E-12	9.68E-15	1.12E-13	4.45E-14	4.53E-14	6.06E-11	6.08E-11
Benzofluoranthene	2.02E-14	2.05E-14	1.01E-15	6.39E-15	2.91E-12	2.95E-12	1.87E-15	3.57E-14	4.31E-15	1.91E-14	1.94E-11	1.95E-11
Benzofluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	1.49E-15	2.47E-15	1.04E-16	2.26E-16	2.12E-13	2.16E-13	1.38E-16	4.30E-15	4.44E-16	6.77E-16	1.42E-12	1.42E-12
Dibenzofluoranthracene	1.29E-12	1.06E-12	5.62E-14	1.11E-12	1.94E-10	1.97E-10	1.20E-13	1.85E-12	2.40E-13	3.34E-12	1.29E-09	1.30E-09
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	5.34E-11	ND	ND	5.34E-11	ND	ND	2.71E-10	ND	ND	2.71E-10
Indeno(1,2,3-cd)pyrene	1.55E-13	6.55E-14	8.41E-15	2.52E-13	1.22E-11	1.27E-11	1.44E-14	1.14E-13	3.59E-14	7.55E-13	8.16E-11	8.25E-11
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total Risk	2.15E-12	1.91E-12	6.53E-11	3.08E-12	3.31E-10	4.07E-10	2.00E-13	9.00E-12	2.79E-10	8.97E-12	2.21E-09	2.51E-09

Notes

ND - Not determined because this compound is not considered carcinogenic by this route of exposure.

NA - Not applicable. This compound is not assumed to be emitted by this set of operating conditions.

Table 5-29
Summary of Potential Hazard Indices for Natural Gas High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Relubrication Project
Lorneville, New Brunswick

Compound	Child									
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	HI
Mercury, elemental	NC	NC	2.56E-12	NC	NC	NC	NC	NC	NC	2.56E-12
Mercury, methyl	6.59E-10	2.59E-09	NC	3.99E-10	1.76E-11	3.15E-10	1.55E-12	1.70E-12	3.86E-14	3.65E-09
Mercury, divalent	3.70E-08	1.02E-08	1.67E-10	5.16E-09	3.89E-09	5.21E-08	6.19E-10	6.38E-10	1.39E-11	5.25E-08
1,4-Dichlorobenzene	1.10E-12	2.29E-09	5.70E-10	5.89E-09	6.75E-14	1.78E-12	3.32E-15	1.37E-12	8.50E-14	8.75E-09
Acenaphthene	6.76E-13	1.83E-11	3.25E-12	5.20E-11	1.59E-14	2.81E-13	1.97E-16	8.11E-14	5.80E-15	7.42E-11
Acenaphthylene	1.13E-09	2.60E-08	3.15E-12	2.36E-09	5.95E-10	1.01E-08	1.64E-12	6.77E-10	6.84E-11	2.95E-08
Anthracene	1.65E-12	5.72E-12	8.66E-13	5.82E-12	7.02E-14	1.09E-12	8.95E-16	3.69E-13	2.38E-14	1.41E-11
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	2.68E-12	2.75E-07	1.34E-07	3.44E-08	5.36E-14	1.42E-12	2.06E-15	8.52E-13	6.96E-14	4.43E-07
Benzo(a)anthracene	1.77E-10	3.26E-10	1.21E-11	7.28E-11	1.02E-10	1.47E-09	1.19E-12	4.90E-10	3.13E-11	5.88E-10
Benzo(a)pyrene	7.54E-11	1.81E-10	7.90E-12	2.08E-10	5.06E-10	8.55E-09	1.42E-12	5.86E-10	5.69E-11	4.72E-10
Benzo(b)fluoranthene	1.65E-10	1.67E-10	1.21E-11	8.88E-11	4.18E-10	6.38E-09	3.66E-12	1.52E-09	1.03E-10	4.32E-10
Benzo(k)fluoranthene	3.98E-10	6.12E-10	1.18E-11	3.82E-10	1.33E-09	2.10E-08	8.81E-12	3.64E-09	2.63E-10	1.40E-09
Benzo(g,h,i)perylene	7.55E-10	1.74E-08	3.15E-11	6.70E-10	2.99E-10	5.02E-09	1.09E-12	4.52E-10	4.17E-11	1.88E-08
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	2.46E-10	6.67E-10	1.21E-11	1.35E-10	2.20E-10	3.36E-09	1.90E-12	7.82E-10	5.35E-11	1.06E-09
Dibenzo(a,h)anthracene	2.05E-10	2.84E-10	6.61E-12	5.97E-10	6.02E-09	1.04E-07	1.01E-11	4.18E-09	5.66E-10	1.09E-09
Fluoranthene	7.57E-11	1.80E-10	8.11E-12	2.89E-11	8.80E-12	1.19E-10	1.32E-13	5.43E-11	3.26E-12	2.93E-10
Fluorene	1.12E-11	2.57E-09	7.58E-12	3.34E-11	1.16E-12	1.98E-11	2.47E-15	1.02E-12	1.10E-13	2.62E-09
Formaldehyde	2.81E-13	1.05E-07	4.06E-08	3.61E-08	9.79E-16	2.61E-14	3.76E-17	1.56E-14	1.27E-15	1.82E-07
Indeno(1,2,3-cd)pyrene	2.40E-10	1.72E-10	9.89E-12	1.32E-09	5.66E-08	9.93E-07	2.75E-11	1.14E-08	4.15E-09	1.74E-09
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	5.55E-11	1.41E-08	4.13E-09	3.37E-08	1.10E-12	2.46E-11	2.65E-14	1.09E-11	8.78E-13	5.20E-08
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	1.35E-12	2.04E-09	4.61E-10	2.00E-10	2.69E-13	6.70E-12	9.91E-15	4.09E-12	3.28E-13	2.70E-09
Pyrene	4.19E-10	9.63E-09	1.75E-11	8.72E-10	2.21E-10	3.75E-09	6.06E-13	2.51E-10	2.53E-11	1.09E-08
Toluene	1.42E-13	6.40E-09	2.94E-10	1.31E-09	4.61E-15	1.21E-13	1.76E-16	7.25E-14	5.91E-15	8.01E-09
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total HI	4.16E-08	1.59E-07	1.80E-07	1.24E-07	7.02E-08	1.21E-06	6.79E-10	2.46E-08	5.38E-09	8.21E-07

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-29

Summary of Potential Hazard Indices for Natural Gas High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Adult									
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	HI
Mercury, elemental	NC	NC	2.19E-12	NC	NC	NC	NC	NC	NC	2.19E-12
Mercury, methyl	1.22E-11	9.01E-10	NC	2.36E-10	3.41E-11	1.38E-10	2.06E-12	2.09E-12	4.45E-14	1.15E-09
Mercury, divalent	6.86E-10	3.55E-09	1.42E-10	3.04E-09	7.53E-09	2.29E-08	7.71E-10	7.83E-10	1.60E-11	7.43E-09
1,4-Dichlorobenzene	2.04E-14	7.97E-10	4.87E-10	3.28E-09	1.31E-13	7.72E-13	4.12E-15	1.68E-12	9.81E-14	4.57E-09
Acenaphthene	1.25E-14	6.39E-12	2.78E-12	2.91E-11	3.08E-14	1.24E-13	2.45E-16	9.95E-14	6.69E-15	3.83E-11
Acenaphthylene	2.10E-11	9.06E-09	2.69E-12	1.41E-09	1.15E-09	4.45E-09	2.04E-12	8.30E-10	7.90E-11	1.05E-08
Anthracene	3.06E-14	1.99E-12	7.40E-13	3.48E-12	1.36E-13	4.80E-13	1.11E-15	4.53E-13	2.74E-14	6.24E-12
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	4.97E-14	9.57E-08	1.14E-07	1.95E-08	1.04E-13	6.26E-13	2.57E-15	1.05E-12	8.03E-14	2.29E-07
Benzof(a)anthracene	3.29E-12	1.14E-10	1.04E-11	4.35E-11	1.98E-10	6.46E-10	1.48E-12	6.02E-10	3.61E-11	1.71E-10
Benzof(a)pyrene	1.40E-12	6.30E-11	6.75E-12	1.25E-12	9.80E-10	3.76E-09	1.77E-12	7.19E-10	6.57E-11	1.96E-10
Benzof(b)fluoranthene	3.06E-12	5.81E-11	1.03E-11	5.32E-11	8.10E-10	2.81E-09	4.56E-12	1.86E-09	1.19E-10	1.25E-10
Benzof(k)fluoranthene	7.39E-12	2.13E-10	1.01E-11	2.29E-10	2.57E-09	9.23E-09	1.10E-11	4.47E-09	3.03E-10	4.59E-10
Benzof(g,h,i)perylene	1.40E-11	6.05E-09	2.69E-11	4.01E-10	5.80E-10	2.21E-09	1.36E-12	5.55E-10	4.81E-11	6.49E-09
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	4.58E-12	2.32E-10	1.03E-11	8.08E-11	4.28E-10	1.48E-09	2.36E-12	9.59E-10	6.17E-11	3.28E-10
Dibenzo(a,h)anthracene	3.81E-12	9.88E-11	5.65E-12	3.58E-10	1.17E-08	4.56E-08	1.26E-11	5.11E-09	6.54E-10	4.67E-10
Fluoranthene	1.41E-12	6.27E-11	6.93E-12	1.72E-11	1.70E-11	5.24E-11	1.64E-13	6.67E-11	3.76E-12	8.82E-11
Fluorene	2.07E-13	8.95E-10	6.46E-12	2.00E-11	2.24E-12	8.72E-12	3.08E-15	1.25E-12	1.27E-13	9.22E-10
Formaldehyde	5.21E-15	3.65E-08	3.47E-08	2.05E-08	1.90E-15	1.15E-14	4.68E-17	1.91E-14	1.47E-15	9.18E-08
Indeno(1,2,3-cd)pyrene	4.45E-12	5.98E-11	8.45E-12	7.91E-10	1.10E-07	4.37E-07	3.42E-11	1.39E-08	4.79E-09	8.64E-10
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	1.03E-12	4.90E-09	3.53E-09	1.90E-08	2.14E-12	1.08E-11	3.30E-14	1.34E-11	1.01E-12	2.74E-08
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	2.51E-14	7.09E-10	3.94E-10	1.20E-10	5.21E-13	2.95E-12	1.23E-14	5.02E-12	3.79E-13	1.22E-09
Pyrene	7.78E-12	3.35E-09	1.49E-11	5.23E-10	4.27E-10	1.65E-09	7.54E-13	3.07E-10	2.92E-11	3.90E-09
Toluene	2.64E-15	2.23E-09	2.51E-10	7.36E-10	8.93E-15	5.34E-14	2.19E-16	8.89E-14	6.82E-15	3.22E-09
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total HI	7.72E-10	1.66E-07	1.54E-07	7.06E-08	1.36E-07	5.31E-07	8.45E-10	3.02E-08	6.20E-09	3.91E-07

Notes:

NC - Not calculated. This compound is not assumed to be present in this medium.

!IA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-30
Summary of Potential Excess Lifetime Cancer Risk for Natural Gas High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Child										Risk
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork		
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
1,4-Dichlorobenzene	5.21E-16	1.08E-12	2.68E-13	2.78E-12	3.19E-17	8.29E-16	1.56E-18	6.45E-16	4.01E-17	4.13E-12	
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Benzene	2.00E-17	2.05E-12	5.64E-13	2.57E-13	3.99E-19	1.08E-17	1.54E-20	6.36E-18	5.19E-19	2.87E-12	
Benz(a)anthracene	6.41E-14	1.24E-13	5.16E-15	2.79E-14	3.85E-14	5.59E-13	4.30E-16	1.77E-13	1.14E-14	2.21E-13	
Benz(b)pyrene	2.77E-13	6.99E-13	3.36E-14	8.19E-13	1.98E-12	3.36E-11	5.21E-15	2.15E-12	2.15E-13	1.83E-12	
Benz(c)fluoranthene	5.98E-14	6.41E-14	5.15E-15	3.47E-14	1.60E-13	2.47E-12	1.33E-15	5.52E-13	3.80E-14	1.64E-13	
Benz(k)fluoranthene	1.19E-14	2.05E-14	5.01E-16	1.48E-14	4.89E-14	7.92E-13	2.64E-16	1.09E-13	8.34E-15	4.77E-14	
Benz(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Chrysene	8.55E-16	2.47E-15	5.13E-17	5.18E-16	8.27E-16	1.28E-14	6.60E-18	2.71E-15	1.89E-16	3.90E-15	
Dibenz(a,h)anthracene	7.10E-13	1.06E-12	2.81E-14	2.35E-12	2.35E-11	4.07E-10	3.50E-14	1.44E-11	2.12E-12	4.15E-12	
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Formaldehyde	ND	ND	3.13E-11	ND	ND	ND	ND	ND	ND	ND	
Indeno(1,2,3-cd)pyrene	8.57E-14	6.55E-14	4.21E-15	5.20E-13	2.23E-11	3.91E-10	9.82E-15	4.05E-12	1.61E-12	6.75E-13	
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Total Risk	1.21E-12	1.91E-12	3.22E-11	6.81E-12	4.80E-11	8.35E-10	5.20E-14	2.15E-11	4.00E-12	4.54E-11	

Notes:

ND - Not determined because this compound is not considered carcinogenic by this route of exposure
NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

Table 5-30
Summary of Potential Excess Lifetime Cancer Risk for Natural Gas High Load Conditions
Farmer at the Northeast Farm
Human Health Risk Assessment
Coleston Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Adult									
	Soil	Water	Air	Produce	Beef	Milk	Poultry	Eggs	Pork	Risk
Mercury, elemental	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, methyl	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mercury, divalent	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	4.84E-17	1.89E-12	1.15E-12	7.75E-12	3.09E-16	1.82E-15	9.73E-18	3.96E-15	2.31E-16	1.08E-11
Acenaphthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	1.85E-18	3.57E-12	2.41E-12	7.28E-13	3.87E-18	2.34E-17	9.57E-20	3.90E-17	2.99E-18	6.71E-12
Benz(a)anthracene	5.96E-15	2.16E-13	2.20E-14	8.35E-14	3.73E-13	1.23E-12	2.67E-15	1.09E-12	6.57E-14	3.27E-13
Benz(a)pyrene	2.57E-14	1.22E-12	1.44E-13	2.46E-12	1.92E-11	7.38E-11	3.24E-14	1.32E-11	1.24E-12	3.84E-12
Benz(b)fluoranthene	5.56E-15	1.12E-13	2.20E-14	1.04E-13	1.55E-12	5.43E-12	8.28E-15	3.38E-12	2.19E-13	2.43E-13
Benz(k)fluoranthene	1.11E-15	3.57E-14	2.14E-15	4.43E-14	4.74E-13	1.74E-12	1.64E-15	6.69E-13	4.81E-14	8.33E-14
Benz(g,h,i)perylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cadmium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chromium VI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chrysene	7.94E-17	4.30E-15	2.19E-16	1.55E-15	8.01E-15	2.81E-14	4.10E-17	1.66E-14	1.09E-15	6.15E-15
Dibenz(a,h)anthracene	6.59E-14	1.85E-12	1.20E-13	7.06E-12	2.28E-10	8.94E-10	2.17E-13	8.84E-11	1.22E-11	9.09E-12
Fluoranthene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Formaldehyde	ND	ND	1.34E-10	ND	ND	ND	ND	ND	ND	1.34E-10
Indeno(1,2,3-cd)pyrene	7.96E-15	1.14E-13	1.80E-14	1.56E-12	2.16E-10	8.60E-10	6.10E-14	2.49E-11	9.29E-12	1.70E-12
Lead	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenanthrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Pyrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vanadium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total Risk	1.12E-13	9.00E-12	1.38E-10	1.98E-11	4.65E-10	1.84E-09	3.24E-13	1.32E-10	2.31E-11	1.67E-10

Notes:
ND - Not determined because this compound is not considered carcinogenic by this route of exposure.
NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions

Table 5-31¹
Comparison of Cancer Incidence Rates
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Comparison of cancer incidence rates per 100,000 population, by gender, Canada², New Brunswick², Health Region 2³ and Health Region 2 areas³
(These rates are for reference comparison only. Rates for Canada and New Brunswick are based on estimated age-standardized rates for 2001.
Rates for R2, CCA, SJA, SJC and SA are based on an average annual crude rate – calculated on incident cases 1989 – 1998 using 1993 population to calculate the rate)

Calculate the rate)		Rate per 100,000										Rate per 100,000					
		Canada	NB	R2	CCA	SJA	SJC	SA	Females	Canada	NB	R2	CCA	SJA	SJC	SA	
Males																	
All Cancers		444	499	515	533	352	635	503	All Cancers	344	353	434	468	311	513	406	
Prostate		118	154	146	147	103	176	154	Breast	105	108	119	124	95	134	122	
Lung		77	95	107	113	63	139	104	Lung	47	50	59	52	37	79	52	
Colorectal		59	61	59	47	41	77	55	Colorectal	38	44	60	81	42	69	46	
Bladder		22	25	33	46	20	37	35	Body of Uterus	18	16	22	24	16	26	16	
Non-Hodgkin's Lymphoma		21	21	20	21	17	21	21	Non-Hodgkin's Lymphoma	15	19	16	22	9	20	13	
Kidney		16	17	17	24	11	22	6	Ovary	13	11	16	15	10	20	22	
Oral		13	10	14	14	8	20	9	Thyroid	9	6	5	5	5	6	4	
Leukemia		13	13	11	11	8	13	16	Pancreas	8	10	11	10	8	14	10	
Stomach		11	14	12	11	8	17	10	Cervix	8	8	10	7	7	13	10	
Pancreas		9	12	11	13	8	14	11	Kidney	8	10	12	13	9	14	13	
Brain		8	7	6	6	6	7	4	Leukemia	8	7	8	11	6	9	3	
Larynx		6	6	9	8	3	13	12	Bladder	6	7	14	10	11	17	12	
Multiple Myeloma		6	5	6	8	5	6	7	Brain	6	5	6	9	6	6	7	
									Oral	5	4	7	7	4	9	4	
									Stomach	5	6	6	4	3	9	7	
									Multiple Myeloma	4	4	5	7	3	5	5	

¹ Table 5-31 has been extracted from "Health Status Report 2001" prepared by Atlantic Health Sciences Corporation, October 2001.

² Canada totals include provincial and territorial estimates. Note: Rates for prostate cancer were estimated based on data years 1980-1989. Rates exclude non-melanoma skin cancer (ICD-9 173) and are adjusted to the age distribution of the 1991 Canadian population. Due to changes and improvements in source data and in methodology, caution is needed if the 2001 estimates are compared to previously published estimates. These estimates may vary from actual figures.

Source: Cancer Bureau, CCDPC, Health Canada; National Cancer Institute of Canada: Canadian Cancer Statistics 2001, Toronto, Canada, 2001
³ R2=New Brunswick Health Region 2; CCA=Charlotte County area; SJA=Saint John area; SJC=Saint John City; SA=Sussex area. Source: Vital Statistics, Health and Community Services, New Brunswick – special tabulations, 2001.

Table 5-32¹

Comparison of Cancer Mortality Rates
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Comparison of cancer mortality rates per 100,000 population, by gender, Canada², New Brunswick², Health Region 2³ and Health Region 2 areas³

(These rates are for reference comparison only. Mortality rates for Canada and New Brunswick are based on estimated age-standardized rates of deaths from cancer for 2001. Rates for R2, CCA, SJA, SJC and SA are based on an average annual crude death rate – calculated on deaths from 1989 – 1998 using 1993 population to calculate the rate)

1995 = 1990 using 1995 population to calculate the rate)															Mortality rate per 100,000							Mortality rate per 100,000						
Males	Canada	NB	R2	CCA	SJA	SJC	SA	Females	Canada	NB	R2	CCA	SJA	SJC	SA													
All Cancers	227	253	247	286	153	304	244	All Cancers	151	158	207	228	128	259	183													
Lung	69	89	89	99	49	115	91	Lung	37	32	48	33	31	68	38													
Prostate	30	32	25	31	14	30	25	Breast	27	26	36	39	25	46	26													
Colorectal	22	16	17	21	8	21	22	Colorectal	14	12	19	27	11	21	25													
Pancreas	9	11	10	9	7	13	9	Pancreas	8	9	11	12	6	14	7													
Non-Hodgkin's Lymphoma	9	11	10	12	7	11	7	Ovary																				
Leukemia	8	6	8	10	5	9	9	Non-Hodgkin's Lymphoma	8	7	11	9	7	13	14													
Stomach	7	10	8	9	8	9	4	Brain	4	4	4	5	3	5	3													
Bladder	7	6	8	7	4	9	12	Leukemia	4	3	5	6	4	5	4													
Kidney	6	9	6	8	6	7	4	Stomach	3	3	5	1	1	8	8													
Oral	5	4	4	9	3	5	0	Body of Uteris	3	4	4	4	1	5	6													
Brain	5	5	5	8	3	6	4	Multiple Myeloma	3	2	3	7	1	2	4													
Multiple Myeloma	4	5	5	6	3	6	7	Kidney	3	5	5	7	3	6	4													
Larynx	3	4	3	3	1	5	2	Oral	2	-	2	2	1	3	3													
								Cervix	2	1	3	4	2	5	3													
								Bladder	2	3	4	4	4	4	3													

- Estimated age-standardized incidence rate is less than 0.5 per 100,000

¹Table 5-32 has been extracted from "Health Status Report 2001" prepared by Atlantic Health Sciences Corporation, October 2001.

²Canada totals include provincial and territorial estimates. Note: Rates adjusted to the age distribution of the 1991 Canadian population. Due to changes and improvements in source data and in methodology, caution is needed if the 2001 estimates are compared to previously published estimates. These estimates may vary from actual figures.

Source: Cancer Bureau, CCDPC, Health Canada; National Cancer Institute of Canada: Canadian Cancer Statistics 2001, Toronto, Canada, 2001

³R2=New Brunswick Health Region 2, CCA=Charlotte County area; SJA=Saint John area; SJC=Saint John City; SA=Sussex area. Source: Vital Statistics, Health and Community Services, New Brunswick – special tabulations, 2001.

03/29/2002

Table 5-33¹
Rate of hospital discharges per 100,000 population for selected chronic conditions, by age group, 1997/1998 and 1999/2000, New Brunswick and New Brunswick Health Region 2
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

	Fiscal Year	Age Category						All ages
		0 - 14	15 - 29	30 - 44	45 - 59	60 - 75	75 +	
ASTHMA ^a								
Region 2	1997/1998	398.7	64.8	76.2	63.8	138.5	72.1	142.7
	1999/2000	283.6	77.2	47.1	89.5	82.4	130.7	116.8
New Brunswick	1997/1998	552.6	128.9	120.1	128.4	278.2	320.7	234.3
	1999/2000	464.7	103.3	91.0	128.4	210.8	257.3	192.4

¹Table 5-33 has been extracted from "Health Status Report 2001" prepared by Atlantic Health Sciences Corporation, October 2001.

Source: Discharge Abstract Database, NB Department of Health and Wellness, special tabulations, 2001

^aIncludes ICD-9 CM categories (4930 - 49399)

Table 5-34¹

Selected Health Statistics for Canada and the Province of New Brunswick
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

Health Statistic	Canada	New Brunswick
Rate of Emergency Department Visits/1000 population, 1995-1996	433.1	190.9
Total Health Expenditures, 1996		
\$ per capita	2,513	2,371
% of GDP	9.2	10.8
Type of Cigarette Smoker (age 12+) and Usage, 1996 and 1997		
Current Smoker (%)	28	28
Former Smoker (%)	29	30
Never Smoked (%)	44	42
Average No. of Cigarettes Daily	17	18
Prevalance of Asthma (% of total population; age 12+), 1996-1997	7	6
Estimated Rates ³ of New Cases of Cancer and Deaths per 100,000 Population, 1998		
New Cases, 1998		
Male	501	532
Female	346	335
Deaths, 1998		
Male	232	255
Female	151	158
Rate of Hospital Separations for Respiratory Diseases per 100,000 Population (1995-1996)	1060	2000
Death Rates per 100,000 Population (age-standardized) (1996)		
All Respiratory Diseases	58	59
Lung Cancer	49	55
Potential Years of Life Lost per 100,000 Population (age-standardized), 1996		
Neoplasms	1098	113
Respiratory	1126	77

Notes:

¹Data have been extracted from various tables in Statistical Report on the Health of Canadians (Health Canada, 1999)²NR = not reported³Excluding non-melanoma skin cancer. Provincial data are age-standardized to the 1991 Canadian population.

Table 5-35

Reported Concentration Averaging and Lag Time Resulting in Highest Statistical Association
Between Air Pollution and Mortality

Human Health Risk Assessment

Coleson Cove Generating Station Refurbishment Project

Lorneville, New Brunswick

City	CO Average (days)	CO Lag (days)	NO ₂ Average (days)	NO ₂ Lag (days)	SO ₂ Average (days)	SO ₂ Lag (days)	Ozone Average (days)	Ozone Lag (days)
Quebec	2	0	2	0	2	1	1	0
Montreal	3	0	3	0	3	0	2	0
Ottawa	2	0	1	1	3	0	1	0
Toronto	1	1	2	0	2	1	2	0
Hamilton	2	0	1	0	2	0	1	0
London	2	1	3	0	1	1	1	1
Windsor	3	2	3	2	2	0	1	2
Winnipeg	2	0	2	1	3	2	1	0
Edmonton	3	2	1	2	2	0	1	1
Calgary	2	0	2	0	2	0	1	0
Vancouver	3	0	2	1	3	0	1	0

Table 5-36

Comparison of Concentration Metrics and Lag Times Identified for Ozone by Canadian Researchers

Human Health Risk Assessment

Coleson Cove Generating Station Refurbishment Project

Lorneville, New Brunswick

Health Outcome	Concentration Metric/Lag Time	Citation
Cardiac Hospitalizations	3-day 8-hour average/ 2 days	Burnett et al. (1997a)
Cardiac Hospitalizations	24-hour average/ 2 days	Burnett et al. (1997b)
Cardiac Hospitalizations	1-day average/ 6 days	Stieb et al. (2000)
Respiratory Hospitalizations	1-hour maxima & 8-hour maxima/ 1 day	Delfino et al. (1997)
Respiratory Hospitalizations	3-day 8-hour average/ 1 day	Burnett et al. (1997a)
Respiratory Hospitalizations	1-hour maxima/ 1 day	Burnett et al. (1997b)
Respiratory Hospitalizations	5-day average/ 1 day	Stieb et al. (2000)
Respiratory Hospitalizations	5-day average of 1-hour maxima/ 0 days	Burnett et al. (2001)
Asthma Hospitalizations	1-hour maxima/ 1- and 3-days	Burnett et al. (1994)
Asthma Hospitalizations	1-hour maxima/ 2 days	Stieb et al. (1996)
Asthma Hospitalizations	3-day average/ 1 day	Burnett et al. (1999)
Chronic Obstructive Pulmonary Disease Hospitalizations	3-day average/ 2 days	Burnett et al. (1999)
Mortality	1-day average/ 1 day	Burnett et al. (1998a)
Mortality	1- or 2-day average/ 0, 1 or 2 days*	Burnett et al. (1998b)
Mortality	1-hour maxima/ 2 days	Cakmak et al. (1998)
Mortality	1-hour maxima/ 0 days	Burnett et al. (2000)

*Results vary for each of 11 cities evaluated.

Table 5-37
Comparison of Measured and Predicted Annual Average Concentrations of Metals in Air
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Chemical	Measured Concentration at Saint John, Forest Hills, 1999 (b) ($\mu\text{g}/\text{m}^3$)	Median Concentration Nationwide (1984-93) (a) ($\mu\text{g}/\text{m}^3$)	Halifax/Montreal (a) ($\mu\text{g}/\text{m}^3$)	Predicted Concentration at Saint John, Existing Operation High Load ($\mu\text{g}/\text{m}^3$)	Predicted Concentration at Saint John, Refurbishment Project High Load ($\mu\text{g}/\text{m}^3$)	Predicted Concentration at Saint John, Natural Gas High Load ($\mu\text{g}/\text{m}^3$)
Lead	-0.002	0.052	NM	7.02E-06	7.17E-09	NA
Arsenic	NM	<0.001	NM	4.36E-07	8.43E-08	NA
Nickel	-0.004	0.002	0.02/0.004	2.06E-04	7.63E-05	NA
Vanadium	-0.02	0.005	0.098/0.013	1.35E-03	3.31E-04	NA
Beryllium	NM	NM	NM	1.58E-08	4.85E-09	NA
Chromium	-0.0002	0.002	0.001/0.006	7.13E-07	2.00E-07	NA
Cadmium	NM	<0.002	NM	1.19E-08	2.32E-09	NA
Mercury	NM	0.001	NM	1.04E-07	4.10E-08	2.88E-10

Notes:

NA - Not applicable. This compound is not assumed to be emitted for this set of operating conditions.

NM - Not measured.

(a) Brook, 1997.

(b) Hughes, 2002.

"-" indicates concentration is approximate.

Table 5-38

Comparison of Measured and Estimated Annual Average Concentrations of Principal Air Pollutants
 Human Health Risk Assessment
 Coleson Cove Generating Station Refurbishment Project
 Lorneville, New Brunswick

	St. John Measured Mean Conc. (ug/m3) 1984-1995	Canada Measured Mean Conc. (ug/m3) 1984-1995	St. John Measured Mean Conc. (ug/m3) 1995-2000	St. John Estimated Existing Operation High Load (ug/m3)	St. John Estimated Refurbishment Project Fuel High Load (ug/m3)	St. John Estimated Natural Gas Fuel High Load (ug/m3)
PM10	15 (^{'84-'95}) 15 (^{'88-'94})	11-42 (^{'84-'95}) 18-28 (^{'88-'94})	14 ^a and 22 ^b	0.0205	0.0134	0.0008
PM2.5	8.5 (^{'84-'95})	7-20 (^{'84-'95})	9 ^a	0.0131	0.0085	0.0008
SO ₂	NA	NA	23 ^a and 13 ^c	1.46	0.944	0
NO ₂	NA	NA	9 ^a and 20 ^c	0.388	0.331	0.0841
Source:	Environment Canada/Health Canada (2000)	Environment Canada/Health Canada (2000)	JWEL (2002)	This report	This report	This report

NA: Not analyzed

a Forest Hills

b 110 Charlotte Street

c 189 Prince William Street

Table 5-39
Comparison of Estimated Soil Concentrations to Background Concentrations in Soils
Human Health Risk Assessment
Coleson Cove Generating Station Refurbishment Project
Lorneville, New Brunswick

Compound	Estimated Soil Concentration (mg/kg)			Background Soil Concentration (mg/kg)		Comments
	Heavy Fuel Oil	Orimulsion	Natural Gas	CCME ^a (mg/kg)		
Mercury, methyl	8.42E-06	1.97E-06	2.34E-08	0.01		Lowest of range (0.01-.04mg/kg) (Hg)
Mercury, divalent	4.73E-04	1.11E-04	1.31E-06	0.01		Lowest of range (0.01-.04mg/kg) (Hg)
Arsenic	4.48E-05	4.42E-06	NA	4.80		Lowest of range (4.8-13.6mg/kg) uncontaminated soils (inorganic As)
Beryllium	4.09E-05	6.38E-06	NA	0.55		Reported mean of range (<1-7mg/kg) in Eastern US) ^b
Cadmium	3.15E-06	3.13E-07	NA	0.71		Location not impacted from local point source (rural parkland)
Chromium	5.15E-03	7.35E-04	NA	33		Reported mean of range (1-1000 mg/kg) in Eastern US) (Cr) ^b
Chromium VI	1.40E-05	2.00E-06	NA	33		Reported mean of range (1-1000 mg/kg) in Eastern US) (Cr) ^b
Lead	2.00E-02	1.04E-05	NA	12		Lowest of range (12-25 mg/kg)
Nickel	4.74E-02	8.95E-03	NA	20		Reported mean of range (5-50mg/kg)
Vanadium	2.40E-01	2.99E-02	NA	38		Lowest of range (38-42mg/kg)

^a Primary guidance for background soil concentrations from Canadian Environmental Quality Guidelines (CCME, 1999)

^b Beryllium and Chromium background soil concentrations from Public Health Assessment Guidance Manual (ATSDR, 1992)
NA: Not applicable

6.0 UNCERTAINTY ASSESSMENT

Within any of the four steps of the risk assessment process, assumptions must be made due to a lack of absolute scientific knowledge. Some of the assumptions are supported by considerable scientific evidence, while others have less support. Every assumption introduces some degree of uncertainty into the risk assessment process. Conservative assumptions are made throughout the risk assessment to ensure that the health of local residents is protected. Therefore, when all of the assumptions are combined, it is much more likely that actual risks, if any, are overestimated rather than underestimated.

The assumptions that introduce the greatest amount of uncertainty in this risk assessment are discussed in this section. They are discussed in general terms, because for most of the assumptions there is not enough information to assign a numerical value that can be factored into the calculation of risk.

6.1 Hazard Identification

During the Hazard Identification step, compounds are selected for inclusion in the quantitative risk assessment from a list of all compounds known or expected to be emitted from the facility. Uncertainty is introduced in three principal areas during this step: (1) estimation of emissions; (2) air quality modeling; and (3) selection of compounds for inclusion in the quantitative risk assessment based on emissions and modeling results.

6.1.1 Development of Emissions Estimates

Emissions from the facility were estimated using a number of information sources, data from facilities with similar combustion and air pollution control technology, engineering calculations, and emission factors published in the literature. These emissions estimates represent the foundation upon which this risk assessment is built. Uncertainties associated with emissions estimation can result in uncertainties in all subsequent risk calculations. Because the emissions estimates are critical to the resulting risk estimates, it is important to acknowledge that a level of uncertainty is associated with them.

Emission factors associated with hexavalent chromium (Cr VI) are associated with an additional level of uncertainty. Emission factors for Cr VI were not available for any of the three sets of operating conditions evaluated in the risk assessment. The emission factors for Cr VI used to evaluate each operating scenario were derived using the assumption that Cr VI emissions comprise 29% of total chromium emissions. The Cr VI emission factors used in the risk assessment are, therefore, 29% of the total chromium emission factor. In evaluating chromium speciation in emissions from oil-fired utility generating units, U.S. EPA (1998d) reports that the average fraction of total chromium in oil-fired utility emissions that is Cr VI is 18%. The risk assessment assumes that a larger fraction of total chromium is Cr VI. Therefore, potential risks estimated for Cr VI are likely overestimated.

The emission factor for mercury in the Refurbishment Project - Orimulsion® - High Load Scenario (7.1×10^{-6} g/sec) was derived based on vendor information and estimates of mercury removal efficiency. This emission factor was increased ten-fold (from 7.1×10^{-6} g/sec to 7.1×10^{-5} g/sec) as a conservative measure. Therefore, potential risks estimated for mercury in the Refurbishment Project - Orimulsion® - High Load Scenario are likely overestimated.

6.1.2 Air Quality Modeling

A computerized air dispersion model has been used to estimate the levels of compounds in air following emission from the facility. The dispersion model provides information regarding how particulates and gases emitted from the facility disperse, or spread, after they are released from the stack. Deposition algorithms are applied to the results of the dispersion modeling to estimate deposition rates.

6.1.3 Particle Size Uncertainty

Section 2.3.2 discussed the fact that the particle size distribution data shown in Table 2-6 is derived from U.S. EPA guidance (EPA, 1998a). There is, however, uncertainty inherent in the use of default data that could affect the average deposition rates calculated by the dry and wet deposition models because it is not certain how well the default distribution represents the final performance characteristics.

6.1.4 Selection of Compounds

The compounds quantitatively evaluated in the risk assessment were selected after an examination of the list of compounds potentially emitted from the facility. Of the compounds potentially present in the predicted emissions from the facility, those selected for analysis are believed to be the most toxic, prevalent, and/or persistent in the environment. Based on an assessment of the literature available on this subject, the compounds evaluated in this risk assessment represent the key compounds in combustor emissions.

6.2 Toxicity Assessment

Dose-response values are usually based on limited toxicological data. For this reason, a margin of safety is built into estimates of both carcinogenic and noncarcinogenic risk, and actual risks are lower than those estimated. The two major areas of uncertainty introduced in the dose-response assessment are: (1) animal to human extrapolation; and (2) high to low dose extrapolation. These are discussed in the following subsections.

6.2.1 Animal to Human Extrapolation

Human dose-response values are often extrapolated, or estimated, using the results of animal studies. Extrapolation from animals to humans introduces a great deal of uncertainty in the risk assessment because in most instances, it is not known how differently a human may react to the chemical compared to the animal species used to test the compound. The procedures used to extrapolate from animals to humans involve conservative assumptions and incorporate several

uncertainty factors that overestimate the adverse effects associated with a specific dose. As a result, overestimation of the potential for adverse effects to humans is more likely than underestimation.

6.2.2 High to Low Dose Extrapolation

Predicting potential health effects from the facility emissions requires the use of models to extrapolate the observed health effects from the high doses used in laboratory studies to the anticipated human health effects from low doses experienced in the environment. The models contain conservative assumptions to account for the large degree of uncertainty associated with this extrapolation (especially for potential carcinogens) and therefore, tend to be more likely to overestimate than underestimate the risks.

6.2.3 Use of Surrogate Toxicity Values

In this risk assessment, all chemicals for which there was no governmental toxicity value from any source for one route of exposure were assigned the toxicity value for the other route of exposure as surrogate values. For instance, chemicals for which there was no inhalation toxicity value were evaluated by the use of the oral toxicity value. This use of surrogate values may introduce some uncertainty, but the alternative is to omit them entirely from the risk assessment. It is far more health-protective to assume that these chemicals are as toxic by the inhalation route of exposure as they are by the oral route than to assume that their toxicity is zero.

6.3 Exposure Assessment

During the exposure assessment, exposure point concentrations are estimated and exposure doses calculated. Exposure point concentrations are the estimated concentrations of compounds to which humans may be exposed. Because the facility is not currently operating under the exact conditions evaluated in the risk assessment for any of the three operating scenarios, exposure point concentrations must be estimated using models containing numerous assumptions, such as the amount of compound released from the facility, the dispersion of the compound in air and its fate and transport in the environment, and the location of people potentially exposed to released compounds. Once the concentrations in an environmental medium such as soil, water, or air have been predicted, the calculation of human exposure and dose involves making additional assumptions. The major sources of uncertainty associated with these assumptions are discussed below.

6.3.1 Estimation of Ambient Air Concentrations and Deposition Rates

Maximum emissions of each of the compounds selected for quantitative evaluation in this risk assessment were estimated, as described below. Compound concentrations in ambient air at ground level are derived based on these maximum emission rates and the results of the dispersion modeling. This concentration represents the exposure point concentration to which a person is assumed to be exposed via inhalation. Therefore, uncertainties associated with these two steps of the hazard identification process can result in uncertainties associated with the resulting ambient air concentrations. Deposition rates are derived based on maximum emission

rates and information from applying the appropriate deposition algorithms to the air dispersion modeling results. As was the case with the estimation of ambient air concentrations, uncertainties associated with both the estimation of maximum emission rates and the deposition calculations can result in uncertainties associated with the resulting deposition rates.

6.3.2 Estimation of Surface Water and Sediment Concentrations

The compound concentrations in surface water in the Spruce, Ludgate, and Menzies Lakes complex were estimated using the U.S. EPA model (1998a,b, 1999b, 2000). Assumptions about the amount and rate of soil runoff, the deposition of particles, the rate of compound degradation, and the size of the catchment, or watershed area, are included in the U.S. EPA model. Each assumption has uncertainty associated with it, particularly because input data were based on information for the general area obtained from generally available literature. Estimating surface water and sediment compound concentrations also involves numerous assumptions regarding the fate and transport of compounds, and the hydrology of local water bodies, such as turn-over patterns and flow rates. These assumptions are conservative to provide reasonable assurance that the evaluation of surface water and sediment exposures does not understate actual exposures.

6.3.3 Estimation of Compound Intake from Food

Estimation of potential compound intake in the food consumed by people living in the study area incorporates many assumptions. Conservative estimates are made about the uptake of compounds into root crops, leaf crops, beef, and dairy products. Parameters, such as crop densities, root uptake factors and interception factors for the produce pathway, and soil and crop ingestion rates, and biotransfer factors for the beef and dairy pathways, are often high-end estimates may not represent actual conditions near the facility.

People may be exposed to compounds in the soil through ingestion of crops and inadvertent ingestion of soil. Because the compounds deposited on the soil surface are bound to or mixed with soil particles, conservative assumptions were made concerning the intake of the compounds by receptors. The conservative assumptions were made to provide reasonable assurance that the evaluation of risks from exposure to soil is not understated. Each conservative assumption tends to overestimate, rather than underestimate, potential risks.

Fish in area water bodies may accumulate compounds in their tissues. Accumulation of compounds in fish tissue is estimated using bioconcentration factors (BCFs) and bioaccumulation factors (BAFs) that are estimated from fish studies which may not reflect actual area conditions. The use of BCFs and BAFs introduces uncertainty into the predicted fish tissue concentrations.

6.3.4 Other Potential Exposure Pathways

The risk assessment evaluated the risks posed by ingestion of freshwater fish after an assumed 30 years of deposition of emissions from the Coleson Cove Generating Station into the nearest watershed. This is health-protective, because it assumes that the fish consumed are mature fish that were exposed to plant emissions for their entire lifetime. Marine fish (both finfish and

shellfish) can also be affected by emissions from the plant, but their exposures would be much less than those of freshwater fish due to their large home range and the large dilution capacity of the marine environment. This is true of material deposited onto the Bay of Fundy as well as of material deposited onto coves, bays and estuaries. In accordance with governmental risk assessment guidance, this risk assessment quantitatively evaluates and reports the results of the worst case situation: repeated ingestion of captive freshwater fish from the most highly affected nearby fresh water bodies.

Fish farming in the coastal environmental can also be affected by material emitted from the plant. However, as above, the fish exposure levels that would occur from direct deposition onto the fish farm holding areas would be much less than the exposures possible for the mature captive freshwater fish.

People in the area collect and dry dulse from nearby coastal areas and then consume it. This exposure scenario was not specifically evaluated in the risk assessment, because the produce ingestion pathway evaluated at the worst case potential residential location is far more health-protective. The produce ingestion scenario assumes that back-yard produce is affected by emissions from the plant everyday for their entire growing season. In addition, in subsequent years, it is assumed that produce takes up chemicals of potential concern by root uptake from the soil. The soil has been subjected to deposition of material from the Coleson Cove Generating Station for an assumed period of 30 years. On the other hand, dulse is harvested and dried over a few day period. The maximum time period over which deposition from the facility could occur is a matter of a few days or a week. For this reason, the risks posed by individuals consuming dulse from the coastal area adjacent to the plant would be far less than the risks posed by ingestion of back-yard produce assumed to be grown at the maximally affected location that could theoretically be developed for residential use in the future. This location is directly adjacent to the NB Power property boundary, where deposition of chemicals of potential concern from the plant are estimated to be the highest.

6.3.5 Effects of Fish Consumption Advisories

In 1999, the Chief Medical Officer of Health in New Brunswick issued a freshwater sport fish consumption advisory (CNB, 1999). Due to increasing levels of health concerns about exposures to mercury, the Chief Medical Officer advised that women of childbearing age and children under the age of eight not consume any freshwater fish. Children eight years of age and older, male adults, and women past child-bearing age should restrict their consumption to one meal every two weeks, which is equivalent to an average daily consumption rate of 16.8 g/day assuming a typical fish meal is 227g in size. Consumption of brook trout in excess of 29 cm should be restricted to one meal per week, which is equivalent to an average daily consumption rate of 33.6 g/day. Consumption of brook trout smaller than 29 cm in length was not restricted.

Fish ingestion rates assumed for this risk assessment are high and thus health-protective. In accordance with the advisory, young children and women of child-bearing age should not be consuming any freshwater fish in the study area. This Risk Assessment assumes that women of child-bearing age consume 81.9 grams per day (g/d) and young children consume 11.4 g/d from

the Lakes complex. If the advisory is being heeded, then risks would be lower than the risks presented in this report.

Adult males and women past child-bearing age are assumed in this risk assessment to consume 81.9 g/day of fish from three specific lakes. If the advisory is being heeded, then their consumption should be restricted to 16.8 g/day, which is nearly five times lower than the value assumed for the risk assessment. Again, the risks posed to people who are following the government advisory would be less than the risks presented in this report.

For example, the total HI for the child fisher living at the maximum hypothetical residence in the Existing Condition - Fuel Oil #6 - High Load Scenario is 0.3. Of this total HI, consumption of fish contributes a hazard index of 0.1. Therefore, if children are not assumed to eat fish, the total HI for the child fisher living at the maximum hypothetical residence would decrease to 0.2 for the Existing Condition - Fuel Oil #6 - High Load Scenario. The estimated ELCR for the child fisher in this scenario would decrease from 8×10^{-8} to 7×10^{-8} .

The total HI for the child fisher living in the City of Saint John in the Existing Condition - Fuel Oil #6 - High Load Scenario is 0.2. Of this total HI, fish consumption contributes a hazard index of 0.1. Therefore, if children are not assumed to eat fish, the total HI for the child fisher living in the City of Saint John would decrease to 0.1 for the Existing Condition - Fuel Oil #6 - High Load Scenario.

The estimated ELCR for the child fisher in this scenario would decrease from 8×10^{-8} to 7×10^{-8} . For the Refurbishment Project - Orimulsion® - High Load Scenario, the total hazard index for the child fisher living at the maximum hypothetical residence would decrease from 0.04 to 0.02 and the ELCR would decrease from 8×10^{-9} to 7×10^{-9} . The total hazard index for the child fisher living in the City of Saint John would decrease from 0.03 to 0.01 and the ELCR would decrease from 2×10^{-8} to 1×10^{-8} .

For the Natural Gas - High Load Scenario, the total hazard index for the child fisher living at the maximum hypothetical residence would decrease from 3×10^{-4} to 5×10^{-6} and the ELCR would decrease from 6×10^{-10} to 2×10^{-10} . The total hazard index for the child fisher living in the City of Saint John would decrease from 3×10^{-4} to 1×10^{-6} and the ELCR would decrease from 4×10^{-10} to 8×10^{-11} .

The total HI for a woman of child-bearing age living at the maximum hypothetical residence in the Existing Condition - Fuel Oil #6 - High Load Scenario is 0.24 (see Section 6.4.2). Of this total HI, consumption of fish contributes a hazard index of 0.16. Therefore, if such women are not assumed to eat fish, the total HI would decrease to 0.08 for the Existing Condition - Fuel Oil #6 - High Load Scenario. The estimated ELCR for the woman of child-bearing age in this scenario would decrease from 3×10^{-7} to 2×10^{-7} .

The total HI for the woman of child-bearing age living in the City of Saint John in the Existing Condition - Fuel Oil #6 - High Load Scenario is 0.20. Of this total HI, fish consumption contributes a hazard index of 0.16. Therefore, if such women are not assumed to eat fish, the total HI would

decrease to 0.04 for the Existing Condition - Fuel Oil #6 - High Load Scenario. The estimated ELCR for the woman of child-bearing age in this scenario would decrease from 3×10^{-7} to 2×10^{-7} .

For the Refurbishment Project - Orimulsion® - High Load Scenario, the total hazard index for the woman of child-bearing age living at the maximum hypothetical residence would decrease from 0.04 to 0.009 and the ELCR would decrease from 3×10^{-8} to 2×10^{-8} . The total hazard index for the woman of child-bearing age living in the City of Saint John would decrease from 0.03 to 0.008 and the ELCR would decrease from 7×10^{-8} to 6×10^{-8} .

For the Natural Gas - High Load Scenario, the total hazard index for the woman of child-bearing age living at the maximum hypothetical residence would decrease from 4×10^{-4} to 1×10^{-6} and the ELCR would decrease from 3×10^{-9} to 4×10^{-10} . The total hazard index for the woman of child-bearing age living in the City of Saint John would decrease from 4×10^{-4} to 6×10^{-7} and the ELCR would decrease from 3×10^{-9} to 3×10^{-10} .

6.3.6 Estimation of Exposure Dose

Once the concentrations of the potentially released compounds in water, soil, air, and food have been predicted through modeling, the extent of human exposure must be estimated. This requires making assumptions about the frequency and duration of human exposure to water, soil, air and food.

Uncertainty may be associated with some of the assumptions used to estimate how often exposure occurs. Such assumptions include location, accessibility, and use of an area. With this in mind, the receptor, or person who may potentially be exposed and the location of exposure were defined for this risk assessment. The locations where certain activities were assumed to take place have been purposely selected because chemical concentrations and frequency of exposure are expected to be high (i.e., use of the maximally affected areas). However, actual frequencies of exposure are likely to be much lower than assumed because residents are not likely to stay in one place and may, for instance, work far away or move to another location. In these cases, the person's potential exposure would be reduced, and the health risks discussed here would be overestimated.

6.3.7 Definition of Study Area

The study area was defined as the area within 20 km of the Coleson Cove Generating Station. Risk assessment guidance usually recommends that the study area be defined as the area within 10 km, but the study area was expanded to 20 km to include the City of Saint John. To ensure that air concentrations and deposition rates were not higher at locations more distant than 20 km, additional model runs were for selected receptor locations at 30, 40, and 50 km from the facility in all four directions. The results demonstrate that the air concentrations and deposition rates at locations beyond 20 km are all lower than those within the 20 km study area. Therefore, the risks estimated to any receptor populations beyond 20 km would be less than the risks reported here for receptors within the study area.

6.4 Risk Characterization

The risk of adverse human health effects depends on estimated levels of exposure and dose-response relationships. Two important additional sources of uncertainty are introduced in this phase of the risk assessment: (1) the evaluation of potential exposure to more than one compound; and (2) the presence of subpopulations which may be particularly sensitive.

6.4.1 Risk from Multiple Compounds

Once exposure to and risk from each of the selected compounds is calculated, the total risk posed by a facility is determined by combining the health risk contributed by each compound. For virtually all combinations of compounds potentially released from combustion facilities, there is little or no evidence of interaction. However, in order not to understate the risk, it is assumed that carcinogenic effects of different compounds may be added together.

Synergism is a phenomenon that has been demonstrated in scientific studies in several circumstances in which the toxic effect of two chemicals together exceeds the toxic effects that would be expected if the two chemicals acted independently. For instance, it is known that the adverse effects of cigarette smoking and asbestos exposure and smoking and radon exposure are both greater than the sum of the risks of the two separate exposures. These two pairs of chemical exposures act synergistically.

The opposite effect has also been demonstrated in the toxicology literature. The phenomenon called antagonism defines the situation when the effect of two (or more) chemicals is less than the anticipated effect if one assumed that the chemicals acted independently. For instance it is well documented in the literature that co-exposure to multiple polycyclic aromatic hydrocarbons can result in a lessened toxic response than exposure to any one alone.

Both phenomena have plausible logical explanations because our enzymatic defense systems, while principally optimized to detoxify foreign chemicals, sometimes inadvertently create chemical metabolites that can be toxic. Synergism can occur when exposure to one chemical causes the body to produce more of an enzyme that is optimized to detoxify, but inadvertently causes a second chemical to become more toxic. Antagonism can occur when exposure to one chemical causes the body to produce more of an enzyme that detoxifies both chemicals to which the person is exposed or that blocks the action of an enzyme which can make a second chemical more toxic. Both concepts are widely used by pharmacologists to design drugs that will act therapeutically in a very specific way.

Risk assessments such as this one, do not quantitatively evaluate synergism and antagonism because there are no methods available from any source to do so. Thus, this HRA may be overlooking the synergistic effects between CPCs that might be occurring but which have not yet been discovered by toxicologists. However, it is just as likely that antagonistic effects are occurring between some of the CPCs and antagonism is also not quantitated in this report.

For instance, it is known that exposure to some metals that are not very toxic, such as zinc, which is an essential nutrient, blocks or antagonizes the absorption of toxic metals, like cadmium, by

tying up the specific transport proteins that are responsible for bringing metal atoms into cells. This antagonistic effect would reduce the risk estimates presented here, but the effect was not included in the health risk assessment because there are specific methods approved by governmental agencies to do so.

Similarly, studies of the toxicity of polycyclic aromatic hydrocarbons shows that antagonism between these molecules is far more often observed than is synergism. It has been shown in numerous animal studies that exposure to potentially carcinogenic PAHs in the presence of noncarcinogenic PAHs results in lower toxicity than exposure to the potentially carcinogenic PAHs alone. This has been explained by the noncarcinogenic PAHs inducing the production of enzymes that change the way the body acts on the potentially carcinogenic PAHs, resulting in more total detoxification.

This risk assessment did not quantitate this antagonistic phenomenon and perhaps overestimated risk by not doing so, but there are no methods approved by regulatory agencies to take antagonistic effects into account.

In conclusion, both synergistic effects and antagonistic effects have been documented in the toxicology literature for specific chemical mixtures. In neither case are there any risk assessment methods available to specifically and quantitatively take the phenomena into account. However, it is equally likely that the two phenomena will exist for any given complex mixture of chemicals. Given that the toxicity factors issued by the government agencies intentionally have large safety factors incorporated into them and given that the exposure assessment intentionally overestimates exposures in many ways, as noted elsewhere in this report, the inability of current risk assessment methods to incorporate synergism and antagonism does not introduce significant uncertainty into the health risk assessment results.

6.4.2 Risk to Sensitive Populations

The health risks estimated in the risk characterization generally apply to the hypothetical area resident whose activities and locations were described in the exposure assessment. Some people will always be more sensitive than the average person and, therefore, will be at greater risk. In evaluating a maximally exposed individual, assuming maximum emission rates and many upper-bound exposure assumptions, this risk assessment estimates potential risks which will overstate any actual risks to over 99% of the exposed population. Dose-response values used to calculate risk, in addition, are derived to estimate potential health risks to these sensitive subpopulations.

This risk assessment specifically takes into account the possibility that the developing fetus and children may be more sensitive to air pollution than adults. First, the differences in inhalation rates and the lower body weights are explicitly incorporated into the risk assessment calculations. Second, the tolerable daily intake (TDI) or reference dose (RfD) values that are used to evaluate the toxicity associated with a certain estimated chemical dose level have been derived by governmental health agencies specifically to be protective of sensitive members of the population, which includes the developing fetus, children, the elderly, and people with impaired health status.

The TDIs and RfDs assume that sensitive members of the population are a factor of ten times more sensitive than a healthy adult.

The risk assessment also takes into account the possibility that elderly adults or adults with pre-existing cardiorespiratory disease may be more sensitive to air pollution than normal adults. It does this by assuming that the exposed adult is an active person who spends 100% of their time outdoors breathing the estimated airborne chemical concentrations. In reality, elderly adults and adults with cardiorespiratory diseases will spend only a small fraction of their time outdoors. Since indoor air concentrations of ambient air pollutants are much less inside of buildings than outside, the risk assessment overestimates the exposures to elderly adults and adults with pre-existing diseases. Secondly, as noted above, the TDIs and RfDs that are used in the risk assessment are defined to be protective of elderly adults and adults with cardiorespiratory diseases.

Women of child-bearing age are often considered to be an especially sensitive sub-population. The risk assessment, therefore, includes a conservative evaluation of potential cancer and noncancer risks for this group. The evaluation of potential exposures of women of child-bearing age was done by assuming the same potential exposures as the adult receptors evaluated in the risk assessment, with the exception that women were assumed to have a body weight of 64.2 kg, rather than the body weight of 70 kg assumed for the adult receptors. Therefore, the potential hazard indices and excess lifetime cancer risks for the woman of child-bearing age receptors would be higher than the hazard indices and risks estimated for the adult receptors in the risk assessment by a factor of 70 kg/64.2 kg, or approximately 1.09-fold.

All estimated total hazard indices (summed across pathways and compounds) for the "woman of child-bearing age" receptors are less than 1.0. All estimated excess lifetime cancer risks are less than 1×10^{-5} . This evaluation is conservative because many potential exposure assumptions used in the risk assessment (for example, inhalation rates, drinking water consumption rates, food consumption rates) are average values for males and females. Because women tend to have lower inhalation rates and consumption rates than men, the values used in the risk assessment overestimate potential exposures (and risks) for women of child-bearing age.

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